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SPECIAL PATHOLOGICAL ANATOMY

A TEXT-BOOK

OF

SPECIAL PATHOLOGICAL ANATOMY

BY

ERNST ZIEGLER
PROFESSOR OF PATHOLOGY IN THE UNIVERSITY OF FREIBURG

TRANSLATED AND EDITED
FROM THE EIGHTH GERMAN EDITION
BY
DONALD MAC ALISTER M.A. M.D.
LINACRE LECTURER OF PHYSIC AND TUTOR OF ST. JOHN'S COLLEGE, CAMBRIDGE
AND
HENRY W. CATTELL M.A. M.D.
DEMONSTRATOR OF MORBID ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA

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SECTION IX

THE ALIMENTARY TRACT

CHAPTER LVIII

THE MOUTH AND TONGUE

174. The **inflammations** of the mucous membrane of the mouth resemble in some points the inflammations of the skin, and in other points those of mucous membranes in general.

The slightest degree of inflammation is described as **erythema.** It is characterised by more or less intense redness of the surface, and either rapidly disappears or passes into the more severe form known as **catarrhal stomatitis.** In this form the mucous membrane is intensely red or livid, its secretion is increased, and its epithelium desquamates. Over the mucous surface of the lips, cheeks, and gums the redness and swelling are in general uniformly diffused, on the hard palate they may appear in streaks and patches. The papillae of the tongue are the structures most affected, and give the surface of the organ a rough tuberculated appearance.

When the inflammatory exudation from the vessels is abundant, translucent vesicles or blebs are sometimes formed on the tongue, lips, and cheeks, where the epithelial covering is thicker or tougher than elsewhere, and so prevents the free escape of the exuded liquid to the surface. Here, as in the external skin, the process of vesiculation is always associated with a certain amount of epithelial destruction. In some cases the excretory ducts of the mucous glands, by retention of their secretion, become dilated into little cysts.

In recent acute catarrh of the mouth the catarrhal secretion contains comparatively few cells; in the later stages the proportion becomes increased. The cells are in part extravasated leucocytes, in part desquamated epithelial cells. If the latter remain on the surface they accumulate and form a whitish or discoloured grey or brown coating or 'fur,' which in the case of the tongue sometimes attains considerable thickness. Cracks and fissures often appear on the surface of the lips: these are apt to exude liquid and become covered with crusts, and often give rise to small ulcers.

Catarrhal stomatitis is generally the result of some mechanical or chemical irritation or infection of the buccal mucous membrane: when the irritation is local, like that caused by a carious tooth, the stomatitis is likewise local. In measles a spotty or macular eruption, and in scarlatina a punctate or continuous bright-red eruption, appears on the mucous membrane. Vesicles and pustules make their appearance on it in small-pox, in varicella, in foot-and-mouth disease, in herpes, and in pemphigus, and these pass through the same stages as those of the skin.

Erysipelatous inflammation of the face extending to or beginning in the mucous membrane of the mouth gives rise to dark or livid redness with much swelling, and often to vesiculation also. The tongue is the part that is most swollen, inasmuch as both the mucous membrane and the intermuscular connective tissue become densely infiltrated with liquid and round-cells.

Aphthous stomatitis is distinguished by the appearance on the catarrhal mucous membrane of opaque greyish-white or yellowish-white rounded or elongated patches (*aphthae*). The patches occur singly or in groups, and are most abundant over the tongue, lips, and cheeks; they are rarer on the palate and fauces. They are surrounded by a livid red border, and occasionally coalesce into larger patches or streaks, though these seldom reach any great size.

According to R. FRÄNKEL, true aphthous stomatitis is a croupous inflammation, in which the epithelium dies and disintegrates, while fibrinous masses consisting of a network of glistening hyaline trabeculae or fibrils, and enclosing leucocytes, are deposited on the underlying connective tissue. We might accordingly term this form of inflammation macular or disseminated fibrinous stomatitis. The fibrinous deposits may disappear gradually, but they are usually cast off, being loosened and extruded by the regenerative ingrowth of the epithelium from the margins. As the process does not involve destruction of the connective tissue, no ulcers or scars are in general left by it; but purulent inflammation is sometimes set up in the zone surrounding the aphthous spots. The eruption appears in successive crops, and may thus be kept up for weeks.

Aphthous inflammation is met with chiefly in children who are teething or otherwise subject to inflammatory affections of the mouth; it also occurs in connexion with anginose sore-throat, pneumonia, gastric catarrh, the acute exanthemata, diphtheria, intermittent fever, whooping cough, etc. It is rare in adults, though it has been observed in women during menstruation, pregnancy, and the puerperal period. Whether bacteria take any part in the causation of aphthae is still undetermined.

Diffuse croupous inflammation and diphtheritic or sloughing inflammation are seldom met with in the mucous membrane of the tongue and cheeks; but they are common over the soft palate, fauces, and tonsils (Art. 183).

Corrosive stomatitis is sometimes produced when caustic substances are swallowed; but the inflammation is usually less marked in the mouth than in the pharynx and oesophagus (Arts. 185 and 186).

Ulcerative stomatitis is an affection that always starts from the alveolar margin of the gums. It begins with redness, swelling, and loosening of the gums around the teeth. The alveolar margin thus becomes rounded and prominent, with blunt subconical processes rising up between the teeth: haemorrhage is not uncommon at this stage. In the second stage of the process the margin of the swollen gum becomes discoloured, and the tissue softens and breaks down into a yellowish friable mass. Ulcers are thus formed, which rapidly deepen, the surface being overspread with shreds of softened tissue. The affection may be either unilateral or bilateral, and generally begins about the front teeth. The ulcerative process is apt to extend directly to the contiguous parts of the cheeks and lips, and in some cases works downward till it attacks and destroys the periosteum of the jaw, leading to necrosis of the bone.

The affection is usually acute, seldom chronic; children are especially liable to it, but adults do not escape. It attacks persons who are badly nourished and debilitated by disease, as in cases of tuberculosis, exhausting intestinal discharges, typhoid, diabetes, or scurvy. Poisonous substances which act chemically and mechanically on the gums, such as mercury (mercurial stomatitis), phosphorus, lead, and copper, also give rise to it if they repeatedly gain access to the mouth. The form which is due to chronic phosphorus-poisoning is very apt to extend deeply into the tissues, and so give rise to periostitis and necrosis of the bones of the jaws (Art. 49). In chronic lead-poisoning the gums are of a bluish or dirty-grey colour.

Noma or gangrene of the cheek ('water-canker' or *cancrum oris*) is either an outcome of ulcerative stomatitis, or begins as an independent affection. In the former case the necrotic disintegration of the gums extends rapidly, and the tissue is speedily changed into a pulpy gangrenous or putrid mass. In the other case a livid swelling appears first on the inner surface of the cheek near the corner of the mouth. A patch of greyish-yellow infiltration very soon follows, and this soon breaks down and becomes gangrenous. Sometimes vesicles appear on the affected surface. From the mucous membrane the process spreads to the outer skin, giving rise to a purplish spot on which at times a blister arises. The spot then becomes black, and gangrene sets in. The surrounding tissue is oedematous and greatly swollen. The affection is generally confined to one side. Once the gangrene has begun the destructive process advances rapidly in all directions and sometimes reaches an astonishing extent, the necrotic destruction involving the nose, jaw-bones, zygoma, etc. The usual issue is death. In rare cases the process comes spontaneously to a standstill, and the wound heals by granulation and cicatrisation, resulting in more or less grave disfigurement of the face.

Noma is most frequent between the ages of two and twelve; it is rarely met with earlier or later. It attacks weakly or debilitated children, who are exposed to unwholesome conditions of various kinds. The diseased parts of the skin contain multitudes of bacteria, but their relation to the affection is not yet clearly made out.

Suppurative inflammation of the mucous membrane of the mouth and of the underlying parts may appear at any spot, but is usually limited to the tongue or the gums. In the latter it frequently arises in connexion with carious teeth. The gum becomes red and swollen, and presently pus forms beneath the surface; the swelling is called a **gum-boil** or parulis. Suppurative inflammation of the tongue (**glossitis**) starts from a wound or ulcer, or from some acute inflammatory affection like erysipelas. According to its mode of origin the whole tongue or a limited part of it becomes swollen, and is soon more or less extensively infiltrated with pus. When the abscess so produced is evacuated, repair is effected by cicatrisation.

References on Stomatitis and Noma.

Babes and Zambilovici: Noma *La Roumaine méd.* **1894**
Bohn: *Die Mundkrankheiten d. Kinder* Leipzig **1866**; *Gerhardt's Handb. d. Kinderkrankh.* IV Tübingen **1877–83**
Demme: Foot-and-mouth disease *Bericht d. Kinderspitals* Berne **1882**
Fränkel: Stomatitis aphthosa *V. A.* 113 **1888**
Hirsch: *Historisch-geograph. Path.* III **1886**, trans. by Creighton (New Syd. Soc.) London **1883–86**
Ranke: Aetiology and morbid anatomy of noma *Jahrb. f. Kinderheilk.* **1888**
Schimmelbusch: Noma *D. med. Woch.* **1889**
Seifert and Kahn: *Atlas der Histopathologie d. Mundrachenhöhle* Wiesbaden **1895**
Siegel: Foot-and-mouth disease in man *D. med. Woch.* **1891** and **1894**
Struch: Noma and its micro-organisms *Inaug. Diss.* Göttingen **1872**
Woronichin: Noma *Jahrb. f. Kinderheilk.* **1887**

175. **Syphilis** gives rise in the mouth to primary, secondary, and tertiary lesions. The primary lesions resemble those of the skin (Art. 159), are commonest on the lips, and are accompanied by indolent lymphadenitis.

The secondary manifestations include in the first place **erythema**, which appears in the form of small circumscribed red spots on the cheeks, tongue, and lips, or as a diffuse reddening of the soft palate and tonsils (*angina syphilitica erythematosa*). Within the erythematous patches not infrequently arise sharply-defined erosions or fissures that are red in tint or overlaid with a whitish film. Secondly, **mucous papules** in the shape of small flattened prominences surrounded by a red areola are produced, whose epithelial covering soon turns bluish-white or pearly (*plaques opalines*), making them look like spots that have been cauterised with nitrate of silver. They occur chiefly on the lips, cheeks, and tongue, though they are not infrequent on the soft palate, tonsils, and pharnygeal walls. They either disappear after shedding their epithelial covering, or grow into gland-like granulomatous nodules, that are bright-red and glazed-looking or covered with a whitish film (LANG). As they break down they leave little ulcers whose floors are covered with shreds of necrotic tissue, and at the corners of the mouth, the edges of the tongue, and the fauces, cracks and fissures are thus produced. All these varieties of lesion are apt to appear in great numbers, and here and there the mucous membrane may be closely beset with them.

In the tertiary stage **gummata** are formed, appearing as nodes from the size of a pea to that of a bean or larger, and situated as a rule in the submucosa. At first the nodes are hard, but afterwards they soften and break through, and thus give rise to **ulcers.** Only in rare instances are they re-absorbed without breaking down and rupturing. The edges and floors of the ulcers are infiltrated and covered with necrotic shreds. The ulcers frequently spread widely, heal slowly, and leave scars whose depth and extent vary with the amount of tissue destroyed.

Gummata are rare in the lips, the corners of the mouth, the cheeks, and the gums. They are more frequent in the tongue, the soft palate, the soft parts covering the hard palate, the fauces, the tonsils, and the walls of the pharynx. In these situations they are liable to give rise to serious ulcerative destruction, extending sometimes to the bone, and often producing perforations of the soft and hard palates.

The cicatricial deformity induced as the disease runs its course is often very considerable. The posterior pharyngeal wall and the soft palate sometimes become adherent, and the fauces and pharynx are apt to become greatly constricted. Atrophy at the root of the tongue, manifested by smoothness and thinning of its mucous membrane, and chiefly due to loss of the follicular glands, has by some been regarded as evidence of syphilitic disease; but it occurs in persons who otherwise show no signs of syphilis.

Tuberculosis of the mouth is usually associated with tuberculosis of the face, lungs, and larynx, but it also occurs as a primary affection. When lupus (cutaneous tuberculosis) extends to the buccal mucous membrane it gives rise to red cellular nodes and nodules, whereby the surface acquires a granular appearance; and these presently break down and ulcerate.

Tuberculosis, whether primary or associated with laryngeal and pulmonary phthisis, commonly attacks the tonsils, fauces, and tongue. It gives rise to nodular excrescences, which when abundantly developed render the affected surface rough and tuberculated. In rare cases deep-seated tuberculous nodes are formed in the substance of the part, and subsequently become caseous.

This is most apt to happen in the tongue; and about such a node the muscular substance is generally studded to a considerable depth with large-celled tubercles (Fig. 309 *a*), each surrounded by a zone of small-celled infiltration (*d*).

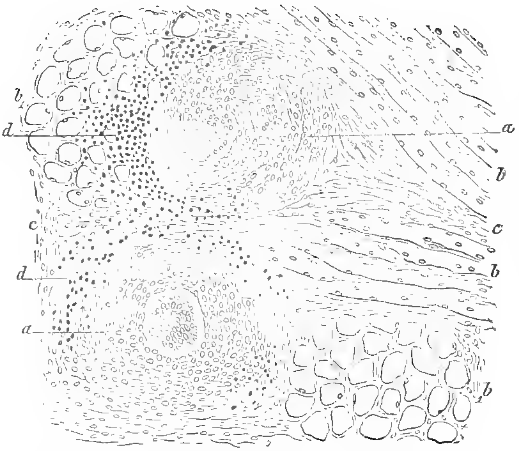

FIG. 309. TUBERCULOSIS OF THE TONGUE.

(*Preparation hardened in alcohol, stained with picro-carmine, and mounted in Canada balsam:* × 80)

a tubercle *b* longitudinal and b_1 transverse section of muscle-fibres *c* connective tissue *d* patches of small-celled infiltration

When a tuberculous node breaks through the surface layers, a tuberculous **ulcer** is produced; this is superficial or deep according to the situation of the node, and in the latter case its margins are infiltrated and more or less undermined.

Actinomycosis, due to the invasion of the ray-fungus or *Actinomyces*, when it appears in man very frequently attacks the tongue and jaws. It is dealt with in the volume on General Pathology.

References on Syphilis and Tuberculosis of the Mouth.

BENDER: Lupus of the mucous membrane *V. f. Derm.* XV **1888**
BUTLIN: *Diseases of the tongue* London **1885**
DUCROT: Tuberculosis of the mouth *Thèse* Paris **1879**
EICHHOFF: Case of wide-spread tuberculosis of the buccal mucous membrane *D. med. Woch.* VII **1881**
FRANK: *Primäre tuberculöse Geschwüre der Zungen- u. Mundschleimhaut* Heidelberg **1880**
GÉLADE: Bucco-pharyngeal tuberculosis *Thèse* Paris **1878**
HANSEMANN: Tuberculosis of the buccal mucous membrane *V. A.* 103 **1886**
LAMBERT: Ulcerations of the tongue in the course of tuberculosis *Thèse* Paris **1876**
LANCEREAUX: *Traité hist. et prat. de la syphilis* Paris **1873**
LANG: *Vorlesungen über Pathol. u. Ther. d. Syph.* II Wiesbaden **1885**
LANGREUTER: Syphilitic strictures of the pharynx *D. A. klin. Med.* XXVII **1881**
LEWIN and HELLER: Smooth atrophy of the papillae of the tongue and its relation to syphilis *V. A.* 138 **1894**
NEDOPIL: Tuberculosis of the tongue *A. f. klin. Chir.* XX **1876**, and *Wiener Klinik* no. 9 **1881**
SCHECH: Syphilitic stricture of the pharynx *D. A. f. klin. Med.* XVII **1876**
SCHLIFEROWITSCH: Tuberculosis of the oral cavity *D. Z. f. Chir.* XXVI **1887**
TROISIER and MÉNÈTRIER: Tuberculous ulcers of the lips *Verneuil's Études sur la tuberculose* III Paris **1891**
WAGNER: Case of tuberculosis of the tongue *A. f. Heilk.* III **1862**

176. The cavity of the mouth is always infested by a multitude of **vegetable microparasites**, which gain entrance to it from without and find in it a fitting soil for their growth. Moulds and yeasts, and schizomycetes or bacteria are all met with; of the latter micrococci and sarcinae as well as bacilli and spirilla occur.

The greater number of these organisms have no pathogenic significance; they are mere saprophytes subsisting on the remains of food and the dead or desquamated epithelium which lie decomposing in the mouth. Where cleanliness is not observed they may occasionally set up putrefactive decomposition and so cause irritation and inflammation. Some varieties of these fungi are stained blue and violet when treated with iodine.

But in addition to these non-pathogenic micro-organisms, pathogenic species very frequently gain access to the mouth. In this connexion it is important to remember that in cases of ulcerative phthisis the bacillus of tuberculosis is always present in the sputum and therefore in the mouth. It appears moreover from numerous observations that the buccal cavity often contains pyogenic micrococci and pneumococci; these occasionally pass into the buccal tissues or into the adjoining parts of the alimentary and respiratory tracts, and there give rise to suppurative or other lesions.

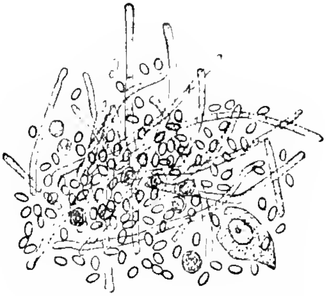

FIG. 310. THRUSH-FILM FROM THE TONGUE.

(*From a man who died of typhoid fever*: × 300)

One of the special parasites of the mouth is the **thrush-fungus** (*Oïdium albicans*), which appears in the form of rounded or oval glistening cells and delicate filaments (Fig. 310). When it first lodges on the mucous membrane it gives rise to the minute slightly-raised white spots commonly called thrush. These may be sparsely scattered or aggregated into clusters, generally on the inner surface of the lips and on the tongue. As they grow and multiply they tend to run together and form a continuous film, which is either white or, when dirty and discoloured, brown, grey, or black. After a time the film is cast off, exposing a reddened and sometimes eroded mucous surface. The thrush-film may reappear on the same spot, and the affection may advance gradually until it reaches the pharynx or even the oesophagus.

The growth and multiplication of the thrush-fungus takes place in the epithelium, which thereby is loosened and degenerates. From the epithelial layer the filaments of the fungus sometimes penetrate the underlying connective tissue, and induce inflammation therein. Young children are especially liable to thrush, the fungus sometimes developing in the perfectly healthy mucous membrane of the new-born infant. Its growth is favoured by the use of sour cow's milk and starchy foods, and by imperfect cleansing of the infant's mouth. Among adults it is generally in cases of debility and wasting from diseases such as typhoid fever, septicaemia, phthisis, etc., that thrush makes its appearance.

References on the Micro-organisms of the Mouth.

BIONDI: The pathogenic micro-organisms of the saliva *Z. f. Hygiene* II **1887**
BUTLIN: The fur of the tongue *St Barth. Hospital Reports* XV London **1879**
DAVID: *Les microbes de la bouche* Paris **1890**
KLEBS: Some mycoses of the buccal cavity *A. f. exp. Path.* V **1876**
MILLER: *Die Mikro-organismen der Mundhöhle* Leipzig **1892**
NETTER: *Streptococcus pyogenes* in healthy saliva *Bulletin méd.* II **1888**
RAPPIN: Bacteria of the mouth *Thèse* Paris **1881**
REUBOLD: Thrush *V. A.* 7 **1854**
VIGNAL: Action of buccal micro-organisms upon certain foods *A. de physiol.* VIII **1886** and X **1887**
WAGNER, E.: Thrush *Jahrb. f. Kinderheilk.* new series Leipzig I **1868**

177. Within the mouth, and in particular over the tongue, desquamation of the epithelium is constantly taking place, and the cellular layers are being renewed by regenerative multiplication. Whenever, from any cause, the growth of new epithelium is increased or its normal desquamation is impeded, whitish deposits are formed on the surface of the mucous membrane, especially on that of the tongue. When the mouth is not kept clean these deposits are often augmented by the remains of food (such as milk), and by the rapidly-growing parasitic fungi which settle in them, and in this way a continuous film or **fur** is produced. This may assume the most various tints, according to the food used, and if the mouth be kept open is apt to dry up into crusts and irregular flakes separated by cracks and fissures.

In cases of chronic irritation, such as is caused by mechanical and chemical lesions (as in tobacco-smoking), by fungus-growths, or by syphilis, the buccal epithelium sometimes undergoes morbid cornification. White streaks and patches are thus produced in the tongue, and these have received various names. The milky or pearly patches of syphilis are called *plaques opalines*. SCHWIMMER proposed for patches having this appearance the name **leucoplakia**, and the term has since been applied to spots due to very different causes, but all characterised by abnormal cornification or **hyperkeratosis** of the epithelium. Others refer to spots and patches characterised by morbid thickening and desquamation of the epithelium, as lingual or buccal leucoma, psoriasis, or ichthyosis. The affections which in the skin are termed **lichen ruber planus** and **acuminatus** form in the mucous membrane silvery-white patches which are isolated or confluent; these result from the coalescence of closely-aggregated miliary nodules, and they consequently feel rough to the touch. In rare cases filiform hairy or bristle-like processes are formed on the tongue, by morbid overgrowth and induration of the epithelium over its papillae. These outgrowths assume, by diffuse staining of the hardened cells (BROSIN), a black or brown colour, and give rise to an affection that has been called **black hairy-tongue** (*glossophytia* or *melanotrichia lingualis*). According to DESSOIS, RAYNAUD, DINKLER, BROSIN, and others, luxuriant colonies of bacteria and mould-fungi often lodge in the neighbourhood of the hardened epithelial filaments; these however are not the cause of the peculiar hyperkeratosis. The condition may be transient or may persist for many years.

Fibrous hyperplasia of the buccal mucous membrane and the contiguous soft parts is due either to some chronic inflammatory process, or to conditions that are congenital or developed from internal causes in early infancy.

Inflammatory hyperplasia is most commonly met with in the gums, giving rise to circumscribed tumour-like thickenings which usually retain for long the aspect and texture of granulation-tissue, and may therefore be described as granulomata. Chronic inflammation of the tongue usually leads to fibrous induration and deformity; but hyperplastic enlargement of the papillae alone (papillary condyloma) is another possible result.

Congenital and infantile hyperplasia affects chiefly the lips (**macrocheilia**) and the tongue (**macroglossia**). The lips are in some cases so thickened as to look like great unwieldy tumours: the tongue may outgrow the capacity of the mouth to hold it, and so presses the teeth outward and protrudes beyond the lips (*prolapsus linguae*, *glossocele*). The protruding part is usually dried up and fissured, and ulcers often form at the points where it is in contact with the teeth. The affection is frequently observed in association with cretinism.

The enlargement of the tongue and lips is due either to an overgrowth of all the constituent tissues (including the muscles) or of the fibrous tissue only, or to the local development of neoplastic tissue and in particular of lymphangioma (Art. 178). The overgrowth of the tissues is either local or general: in the former case isolated nodes are produced.

In the purely fibrous form of hyperplasia the muscle-fibres are generally atrophied. The connective tissue itself is sometimes firm and hard, sometimes cellular or here and there infiltrated with leucocytes. The infiltration is most marked when the protruding portion of the tongue is fissured and ulcerated, and so subject to intercurrent inflammation.

Of the **atrophies** and **degenerations** to which the tissues of the mouth are liable, simple and fatty atrophy and waxy degeneration of the lingual muscles are the most important. They depend on local disorders of nutrition due to inflammatory conditions, or on neurotic disturbance in connexion with affections of the hypoglossal nerve and its nucleus in the medulla oblongata, in consequence of which one-half of the tongue undergoes atrophy (lingual hemiatrophy).

A common form of superficial atrophy affecting the root of the tongue, characterised by smoothness and thinness of its mucous membrane and by disappearance of its glandular follicles, is commonly regarded as a result of syphilitic infection; but as we have already said it is also met with in persons who present no signs whatever of antecedent syphilis.

Amyloid degeneration of the tongue is very rare.

Atrophy of the gums and of the alveolar processes of the jaws is apt to follow upon loss of the teeth, and is especially notable in advanced age.

References on Certain Affections of the Tongue (see also Art. 178).

Artaud: *De l'hémiatrophie de la langue dans le tabes dorsal ataxique* Paris **1885**
Bernhardt: Black hairy-tongue *Unna's Dermatologische Studien* VIII **1888**
Brosin: Black hairy-tongue *Unna's Dermatologische Studien* VII **1888**; *Die schwarze Haarzunge* Leipzig **1888**
Butlin: *Diseases of the tongue* London **1885**
Ciaglinski and **Hewelke**: Black tongue *Z. f. klin. Med.* XXII **1893**
Crocker: Lichen ruber *Monatsh. f. prakt. Derm.* I Hamburg **1882**
Debove: Buccal psoriasis *Thèse* Paris **1873**
Dessois: Black tongue *Thèse* Paris **1878**
Dinkler: Blackness of the tongue and analogous changes in its mucous membrane *V. A.* 118 **1889**
Erb: Hemiatrophy of the tongue *D. A. f. klin. Med.* **1885**; Cicatricial *plaques Münch. med. Woch.* **1892**
Helbing: Muscular macroglossia *Jahrb. f. Kinderheilk* XLI **1896** (with full references)
Hirt: Hemiatrophy of the tongue in a case of tabes *Berl. klin. Woch.* **1885**
Humphry: Prolapse of the tongue *Med.-chir. Trans.* XXXVI London **1853** (with references)
Kaposi: *Art.* Lichen *Eulenburg's Realencyklop.* **1894**
Koch and **Marie**: Hemiatrophy of the tongue *Rev. de méd.* VIII **1888**
Lange: Papilloma of the tongue and tonsils *D. A. f. klin. Med.* L **1893**
Leloir: Buccal leucoplasia *A. de physiol.* X **1887**
Lewin and **Heller**: Smooth atrophy of the root of the tongue *V. A.* 138 **1894**
Maguire: Macroglossia *Journ. of Anat.* XIV **1880**
Mauriac: Lingual and buccal psoriasis *Union méd.* **1873** and **1874**
Morris: Ichthyosis and epithelioma *Lancet* I **1882**
Mourek: Green hairy-tongue *A. f. Derm.* XXIX **1894** (with references)
Nedopil: Lingual psoriasis and carcinoma *A. f. klin. Chir.* XX **1876**
Pel: Hemiatrophy of the tongue with left-sided paralysis of the palate etc. *Berl. klin. Woch.* **1887**
Paster: Macroglossia and macrocheilia *Jahrb. f. Kinderheilk.* new series XVIII **1882**
Raymond and **Artaud**: Hemiatrophy of the tongue *A. de physiol.* **1884**
Schwimmer: Leucoplakia *V. f. Derm.* IV **1877** and V **1878**, *Trans. internat. med. congress* III London **1881**
Touton: Lichen ruber *Berl. klin. Woch.* **1886**
Virchow: *Die krankhaften Geschwülste* III **1868**; Macroglossia *V. A.* 7 **1854**
Wegner: Lymphangioma *A. f. klin. Chir.* XX **1876**

WEIR, R. F.: Ichthyosis of the tongue and vulva *New York Med. Journ.* **1875**
WIEDEMANN: Macroglossia and macrocheilia *Inaug. Diss.* Würzburg **1887**
WILSON: Lichen ruber (cases) *Journal of Cutaneous Medicine* II London **1869**
ZIEGLER: Amyloid tumours of the tongue *V. A.* 65 **1875**

178. The most important of the **tumours** affecting the mouth in early life are the **angiomata** and **lymphangiomata**. Angiomata are met with chiefly about the lips, appearing as dark-red or livid slightly-raised patches. Lymphangiomata occur under or about the tongue, whence they may spread to the soft palate or less often to the cheeks. They also give rise to some of the cases of enlargement of the tongue described as macroglossia. Not infrequently the chief or at least some considerable part of the swollen tongue consists of dilated lymph-vessels; and the entire organ, muscles, mucous membrane, and papillae, is occasionally transformed into a kind of spongy mesh-work, whose spaces contain lymph and whose trabeculae are made up of fibrous tissue interspersed with a varying number of muscular fasciculi. The fibrous tissue may or may not be highly cellular; in the former case it contains patches of lymphadenoid tissue. In other cases the trabeculae contain an extraordinary amount of fat, the tumour being then most fitly described as a **lymphangio-lipoma**. The dilated lymph-spaces are usually of no great size, but sometimes they become distended into globular cysts from the size of a pea to that of a middle-sized cherry (**cystic hygroma**).

Among the other congenital or infantile growths, the chief are teratomata, lipomata, fibromata, myxomata, and sarcomata. These are of various sizes and occur in different places. The teratomata are sometimes autochthonous or due to misplacement of tissue within a single foetus (ARNOLD), sometimes heterochthonous or the result of partial inclusion of a twin embryo. They consist in certain cases only of fat, cartilage, and connective tissue covered with hairy skin; in other cases they include bone, cartilage, fat, nerves, and connective tissue, with glands, cysts, teeth, and even rudimentary organs.

Of the tumours which appear in later life sarcoma and carcinoma are the most notable. **Sarcoma** mainly affects the gums (sarcomatous epulis), rarely appearing elsewhere, and as a rule starts in deeper-lying structures like the periosteum or bone-marrow. It forms rounded tuberous growths, usually somewhat firm in consistence. When it starts from bone it generally includes osseous trabeculae (osteo-sarcoma) and often contains giant-cells (myeloid sarcoma).

Carcinoma attacks the lips, tongue, and gums. It begins as a small nodule, or a circumscribed hard greyish-white infiltration of the mucous membrane. Presently this becomes a palpable node projecting above the surface. The infiltrated tissue ulcerates, and around the ulcer the cancerous infiltration spreads more or less rapidly.

Adenoma of the mucous glands is a rare form of tumour; it appears in the form of isolated nodes and in some cases gives rise to macrocheilia.

References on Tumours of the Mouth and Pharynx.

Arnold: Hairy polypi of the pharynx and mouth *V. A.* 111 **1888**
Arnstein: Macroglossia (cases) *V. A.* 54 **1872**
Bazinsky: Congenital macroglossia, cretinism, and rickets *Henoch's Festschrift* Berlin **1890**
Billroth: *Beiträge zur path. Histologie* Berlin **1858**
Butlin: Glandular tumours of the tongue *Trans. Clin. Soc.* XXIII London **1889**
Esmarch and Kulenkampff: *Die elephantiastischen Formen* Hamburg **1885**
Fraenkel: Macrocheilia due to adenoma of the mucous glands *A. f. klin. Chir.* XLIV
Gies: Macroglossia *A. f. klin. Chir.* XV **1873**
Gosselin: Lipoma of the tongue *Progrès méd.* VIII **1880**, *Paris méd.* no. 20 **1881**
von Langenbeck: *A. f. klin. Chir.* XXI **1878**
Maas: Macroglossia *A. f. klin. Chir.* XIII **1872**
Otto: Case of congenital hairy polypus of the pharynx *V. A.* 115 **1889**
Ranke: Serous cysts of the cheeks *A. f. klin. Chir.* XXII **1878**
Samter: Lymphangioma of the buccal cavity *A. f. klin. Chir.* XLI **1891**
Scheier: Sarcoma of the tongue *Berl. klin. Woch.* **1892** (with references)
Schmidt: Autochthonous teratoma *Inaug. Diss.* Königsberg **1889**
Schuchardt: Hairy polypus of the pharynx *Cent. f. Chir.* **1884**
Seifert and Kahn: *Atlas d. Histopathologie d. Nase u. Mundrachenhöhle* Wiesbaden **1895**
Wegner: Lymphangioma of the tongue *A. f. klin. Chir.* XX **1877**
Winiwarter: Congenital macroglossia and cystic hygroma *A. f. klin. Chir.* XVI **1874**
Yersin: Angioma of the base of the tongue *A. de physiol.* VII **1886**

179. **Ranula** is a cystic formation situated under the tongue close to the fraenum.

According to von Recklinghausen, true or classical ranula is a cystic dilatation of one of the main ducts of the mucous glands of Nuhn and Blandin situated at the tip of the tongue, due to obstruction of the duct by inflammatory changes either within or around it. C. Neumann, on the other hand, maintains that it arises in the ducts of the lingual glands of Bochdalek, resting his opinion chiefly on the fact that ciliated epithelium is sometimes found within the cyst.

The contents of a ranula arising in the glands of the tip of the tongue consist of a viscid transparent mucous or glairy liquid, resembling the white of an egg, and either quite colourless or of a pale-yellow, brown, or reddish tint. It does not contain saliva. The cyst is usually globular or ovoid in shape.

Besides this typical form of ranula, there are other cysts which appear beneath the tongue and are loosely called by the same name. Wharton's duct, leading from the submaxillary gland, may be distended into a cyst. The cyst in this case is usually fusiform, ampullate, or cylindrical, though at times it is more globular in shape. The occlusion of the duct is generally due to inflammation or to the formation of salivary concretions or calculi.

The ducts of the sublingual glands (ducts of Rivini and Bartholin) may also be distended as a result of constriction of their lumen, and form sublingual cysts. Dermoid cysts are met with in the same situation. Finally, congenital cysts of the branchial clefts (congenital cystic hygromata) occasionally protrude under the tongue, and so form spurious ranulae.

Cysts occur in other situations, but much less frequently. Thus they are found at times in the muscular substance of the tongue and in the mucous membrane of its base. Such cysts are usually small, but now and then they attain a very considerable size (Bochdalek, Lotzbeck, Hammerich). They are due to dilatation of the glands that exist at the base and around the root of the tongue.

The mucous glands of the lips may in like manner be transformed into cysts, which vary from the size of a pea to that of a hazel-nut.

According to Virchow, Reubold, Bohn (*Die Mundkrankh. der Kinder* Leipzig **1866**), Denis, Billard, and others, in most new-born infants the mucous membrane of the palate (about the raphé and anteriorly) is more or less beset with white nodules of the size of a millet-seed or a pin's head, which remain unchanged for a long time, or ulcerate and give rise to little sores. These nodules are developed in the second half of foetal life, and are due to an accumulation of epithelial cells within the mucous glands of the hard palate. They might be described as milium or comedones of the mucous membrane.

References on Ranula and other Cysts of the Tongue.

Barker: Dermoid cyst *Trans. Clin. Soc.* XVI London **1883**
Bochdalek: Lingual cysts *Oesterr. Z. f. prakt. Heilk.* XII **1866**
Hammerich: *Ueber Schleimcysten der Zungenwurzel* Würzburg **1877**
Lotzbeck: Lingual cysts *Memorabilien* XV **1870**
Neumann: Ranula *A. f. klin. Chir.* XXXIII **1886**
von Recklinghausen: Ranula *V. A.* 84 **1881** (with references)
Suzanne: Ranula *A. de physiol.* X **1887**

CHAPTER LIX

THE TEETH

180. By far the most important morbid change affecting the teeth is that known as **caries**, a gradually-progressive disintegration of the enamel and dentine.

First of all an opaque white spot (which is sometimes discoloured green or black) appears on the transparent enamel. The colour is due to the fact that the prisms of enamel are loosened and to some extent broken down. Then by degrees the dentine is attacked, and decalcification and loss of substance speedily ensue.

In the advancing margin of the diseased area the dentinal tubules are widened, and in section appear surrounded by bright rings. Microscopic examination reveals the presence within the tubules of micrococci, bacilli, and leptothrix-filaments. According to MILLER, the teeth are rendered susceptible to the action of these bacteria by antecedent acid fermentations (like that producing lactic acid) in the remains of food lodged about and between them, whereby the enamel and dentine are decalcified. SCHLENKER affirms that the enamel-cuticle (Nasmyth's membrane) is first removed by the action of pepsin and acids (such as those of fruit), and then the substance of the tooth is attacked. The enamel may also be eroded or broken through by mechanical injuries. The disintegration of the softened dentine is probably effected by more than one kind of micro-organism (MILLER), as several of the bacteria of the mouth have the power of breaking up albumin or albuminoid substances, or of changing them into soluble forms. Caries never starts from the interior of the tooth.

A very common result of caries is inflammation of the dental pulp or of the alveolar periosteum. The irritants which directly induce it are the bacteria which are present in the disintegrated dentine, and set up septic decomposition therein.

The inflammation of the pulp and periosteum may pass into suppuration. In this case the surface of the gum in the neighbourhood of the diseased tooth is red and swollen (**gum-boil** or *parulis*), and presently suppuration sets in and extends to the tissue of the gum, forming an **alveolar abscess**. This ruptures externally, and if the suppurative process around the root of the tooth continues, an alveolar sinus or fistula is formed.

Sometimes the inflammation extends beyond the region of the root of the tooth, and gives rise to an extensive periostitis of the jaw. In this way large abscesses are sometimes formed, and necrosis of portions of the jaw-bone is occasionally induced.

In chronic non-suppurative inflammation of the exposed pulp and periosteum, granulations, new bone, and new dentine are sometimes produced.

HUTCHINSON (*London Hosp. Reports* II **1865**, *Trans. Path. Soc.* London **1858–59**, *Clinical Surgery* XI London **1878**) first pointed out that the permanent incisors, and especially the upper central incisors, of children suffering from **congenital syphilis** often undergo a peculiar arrest of development. They are either stunted, or as they emerge from the gum their sides converge while the cutting edge is slightly concave. After a time the dentine is exposed and the edge becomes more deeply notched. When the tooth is full-grown it appears pointed or peg-shaped, its apex being truncated by a crescentic notch. The cause of the deformity is said to be a specific alveolar stomatitis during infancy (BÄUMLER: *Ziemssen's Cyclop.* III).

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181. **Tumours** arising from the teeth themselves and composed of dentine are described as **odontomata**. They are formed from the dental pulp during the development of the teeth, and give rise to tuberosities or excrescences on its crown or root.

Tumours of dental tissue formed in later life are described by dental pathologists as **odontinoids** (ULRICH). According as they consist of enamel, dentine, cement, or of a combination of these, they are classified as enameloid, enamelo-dentinoid, dentinoid, dentino-osteoid, or osteoid (SCHLENKER). They are all small, and often to be recognised only by the aid of the magnifying lens. They are flat, round, pear-shaped, or warty in appearance. The three first-named varieties grow from odontoblasts, and arise both from the pulp of the crown and from that of the root, generally in connexion with caries, under metallic fillings, or as a result of periostitis, mechanical injuries, abnormal retention of teeth, or senile degeneration. The osteoid form grows from the pulp or from the periosteum, and is developed from osteoblasts.

Calcoid is the name given by SCHLENKER to a degenerate condition of the pulp, which causes it to crumble in the process of grinding. The calcoid substance is composed of connective tissue, containing mere traces of dentine and cement-like material.

Sarcoma, fibroma, and myxoma are in rare cases developed from the pulp as the tooth is being formed. Such growths, and particularly the sarcomata and fibromata, are however much more commonly derived from the periosteum of the dental socket or alveolar process, from the bone-marrow, or from the gum itself. They arise close to the teeth or actually from their sockets, and are included under the surgical term **epulis**.

Special mention must be made of the **cysts** of the jaws which appear in or about the alveolar ridge and sometimes attain a considerable size. They are unilocular or multilocular. The smaller ones lie hidden in the jaw, the larger protrude through the bone and in some cases reach the size of a man's fist, being covered over with a thin layer of bone or occasionally of fibrous tissue only. Their contents usually consist of a clear mucous or syrupy brownish-coloured liquid, with tablets of cholesterin in some instances. In very rare cases the contents resemble those of certain dermoid cysts (MIKULICZ), namely masses of more or less stratified epithelium with a nacreous lustre. These cysts are of dental origin, and are produced by morbid dilatation of the socket of a mature tooth or by cystic degeneration of the epithelial enamel-germ or of the follicle of a rudimentary tooth. In the latter cases the cysts are lined with cylindrical epithelium. According to the way in which the cyst has arisen, roots of mature teeth sometimes project into its cavity, or it contains rudimentary teeth in various stages of development or mature but generally malformed teeth, sometimes in very large numbers.

FALKSON has described cases of **cystadenoma**, arising from the rudimentary tooth-papillae and taking the form of a multilocular cyst, the cavities of which are lined with cylindrical epithelium and are produced by cystic degeneration of the dental follicles. The growth encloses newly-formed gland-like tubules and acini. P. BRUNS and others have recorded certain rare instances of dental **carcinoma**, in which some of the epithelial cells of the tumour take on the appearance of enamel-germs and produce enamel (CHIBRET).

Renewal of the teeth after the first or deciduous dentition normally takes place but once: in very rare cases however, in young persons, from two up to six **successive dentitions** have been observed. In old age a tertiary dentition sometimes occurs, but the number of authentic instances is not large. Some of the cases described have apparently been due to the re-emergence of buried stumps in the course of senile shrinking of the jaw.

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CHAPTER LX

THE THROAT

182. The **mucous membrane** of the throat (including in the term the soft palate, tonsils, fauces, and pharynx) resembles that of the mouth in structure; it contains however a larger amount of lymphadenoid tissue, which at various points is aggregated into nodules and follicles. In the tonsils especially this tissue is very abundant. STÖHR has shown that lymphoid cells are continually migrating from the lymphadenoid tissue to the free surface, and they often pervade the epithelium in large numbers.

Inflammation of the throat (referred to generally as **angina** or pharyngitis) is due to mechanical, chemical, or thermal irritation, or to some general infective disease like diphtheria, measles, scarlatina, or small-pox. The catarrhal varieties give rise at first to redness and swelling, which may be diffused or disposed in irregular streaks and patches. Then the mucous membrane pours out a mucous or muco-purulent (rarely sanguinolent) secretion, which covers the surface and with the loosened and desquamated epithelium forms a whitish film or deposit upon it.

In some inflammatory affections, such as those which accompany small-pox or herpes labialis, vesicles are formed (*angina vesiculosa*): these speedily rupture and leave small erosions of the epithelial surface.

In inflammation of the soft palate and fauces the **tonsils** seldom or never escape entirely, and in many cases indeed they are involved to an extreme degree (**tonsillitis**, *angina tonsillaris*, *amygdalitis*).

In catarrhal inflammation of the tonsils the surface is covered with secretion, while at the same time migratory cells and desquamated epithelium collect in the crypts, and not infrequently project from their mouths as little plugs (lacunar or follicular tonsillitis). When these plugs are allowed to remain in the crypts they become condensed into greasy masses, and are ultimately transformed into chalky concretions (tonsillar calculi), from the size of a millet-seed to that of a bean, or in rare cases larger still. The accumulated secretion often undergoes putrefactive changes, and then becomes foetid and acts as an irritant on the surrounding tissue.

The inflammatory swelling of the tonsils varies greatly in amount, and manifestly depends on whether the inflammation is merely superficial or affects the entire substance of the organ (parenchymatous tonsillitis).

In **chronic inflammation** of the soft palate and pharynx, the secretion thrown out by the mucous membrane is mucous or muco-purulent, or it consists of greenish-yellow pus which dries into crusts and films that often emit an unpleasant odour. Sometimes the inflammatory secretion is scanty or absent altogether. The mucous membrane of the pharynx is more or less reddened, and is sometimes studded with nodular, ridged, or papillomatous excrescences, whence the affection has been termed **granular pharyngitis** (*pharyngitis hyperplastica, granulosa,* or *papillomatosa*). In other cases the membrane is brownish-red, thin, smooth, and glazed, a condition that is generally called **atrophic pharyngitis.** Atrophy of one part of the mucous membrane may be combined with thickening of another part. The thickening is due primarily to increase of the lymphadenoid tissue of the mucosa, and is accordingly most marked in the region of the pharyngeal tonsil in the naso-pharynx, where it is occasionally so extreme as to fill up the naso-pharyngeal cavity, and to obstruct the posterior nares and the eustachian orifices. Papillomatous thickening of the mucous membrane is also in some instances due to hyperplasia of its connective tissue (ROTH, MÉGEVAND), affecting chiefly the pillars and recesses of the fauces, the lateral walls of the pharynx, and the uvula. The hyperplastic fibrous tissue is usually more or less densely infiltrated with leucocytes. When the hyperplasia is accompanied by the formation of smooth atrophic patches in the epithelium, the connective tissue underlying these patches is likewise atrophic, and the lymphadenoid tissue is reduced in amount.

Sometimes the epithelium overlying the thickened mucous membrane is not perceptibly altered, though it is always more or less infiltrated with leucocytes: over the aggregations of lymphadenoid tissue it is often attenuated and in some parts ulcerated. Along with this the epithelium is apt to be thickened in places, giving rise to whitish patches on the surface.

Unilocular or multilocular **cysts** are now and then formed in the pharyngeal vault. They arise either from cystic degeneration of the so-called pharyngeal bursa, a pouch of mucous membrane directed towards the occipital bone that often persists from the foetal period, from morbid recesses produced in the pharyngeal tonsil, or from the ducts of the pharyngeal glands. The contents are mucoid or resemble atheromatous detritus, and the walls are lined with different varieties of epithelium.

Hypertrophy of the tonsils and follicular glands may result from antecedent inflammation, or appear idiopathically: the latter form is commonest in children. It is due to overgrowth of the lymphadenoid tissue, whereby both tonsils and follicles occasionally attain a considerable size. In cases of leukaemic and pseudoleukaemic enlargement of the lymph-glands (Art. 38), the lymphadenoid tissue of the pharynx is apt to become hyperplastic.

Atrophy of the tonsils results from inflammation, or accompanies senile decay or general marasmus. It depends on diminution of the lymphadenoid tissue. Inflammations are moreover in some cases followed by cicatricial induration and contraction. The crypts of the morbidly-altered tonsils often contain plugs of secretion or calculi.

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183. **Croupous, diphtheritic,** and **gangrenous** inflammations of the soft palate, fauces, tonsils, and pharynx are produced by various forms of injury and infection, but they are most frequently due to diphtheria. Other infections, in particular scarlatina, and less frequently measles, typhoid fever, small-pox, and dysentery, are also capable of inducing them; though it should be remembered that even epidemic diphtheria and scarlatina do not always give rise to severe inflammations of this region.

In epidemic **diphtheria** the inflammatory process chiefly affects the mucous membrane of the soft palate, fauces, tonsils, and pharynx; but it very often extends to the nose, larynx, trachea, and bronchi, and less frequently to the buccal mucous membrane. It is accompanied by swelling of the cervical lymph-glands. In the parts first mentioned it is characterised primarily by the formation of membranous deposits, which on the first day appear as grey opalescent or white spots, and afterwards as thicker dirty greyish-white or yellowish flakes. These overlie it may be the reddened and swollen tonsils, or parts of the soft palate, the uvula, the fauces, and the pharyngeal walls, and occasionally coalesce into bulky membranes of larger size. After solution or ablation of these membranes, a reddened surface is left which only in rare cases is ulcerated, and this as a rule only in the tonsils. The denuded surface may again become covered with new deposits. The false membrane at first adheres with some tenacity to the underlying tissue, both during life and after death, and therefore looks like a superficial slough of the mucous membrane. At times two or three layers can be made out in the membrane even with the unaided eye. These admit of partial separation from one another, especially over the uvula, which is often considerably swollen and as it were swathed in a thick membranous sheath.

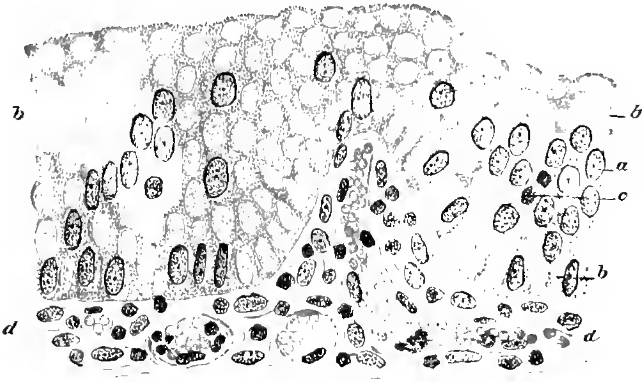

FIG. 311. SUPERFICIAL DIPHTHERITIC NECROSIS OF THE EPITHELIUM OF THE EPIGLOTTIS.

(From a case of diphtheria with croupous pharyngitis: preparation hardened in Müller's fluid, stained with hematoxylin, and mounted in Canada balsam: × 300)

a normal epithelium with well-stained nuclei
b necrotic epithelium with unstained nuclei
c leucocytes in the epithelium
d hyperaemic and infiltrated fibrous tissue

The changes leading to the formation of the whitish spots chiefly concern the epithelium, which by the action of the specific infection becomes to some extent necrotic (Fig. 311 *b*). According to **HEUBNER** the epithelium is moreover at an early stage infiltrated with a liquid exudation, which by and by coagulates, and forces asunder the epithelial cells, especially in the surface layers. The underlying fibrous tissue is at this stage hyperaemic, and shows signs of inflammatory infiltration (Fig. 311 *d*).

The degenerate and infiltrated epithelium is apt speedily to perish by solution and desquamation, but in places where there is no friction the membrane, consisting of fibrin and dead epithelial cells, may remain adherent. Portions of the deeper layers of the epithelium often persist for a long time, while continuously or at intervals liquid and cellular exudations pass from the seat of inflammation to the surface, and there coagulate (HEUBNER).

Sooner or later (from the second to the fifth day) the epithelium at the seats of exudation is entirely or almost entirely lost, and the submucous connective tissue is covered only with a reticular mass of fibrin (Fig. 312 *c*), enclosing in its meshes a few epithelial cells, some of which are unchanged and others degenerate or fused into peculiar flakes. This fibrinous covering extends downwards into the substance of the inflamed mucous membrane, itself often permeated by delicate threads of fibrin (Figs. 312 *d* and 313 *l*). Thus the diphtherial infection induces in the first place an inflammation associated with superficial exfoliation of the epithelium (superficial diphtheritis), and this is followed by fibrinous or **croupous inflammation.**

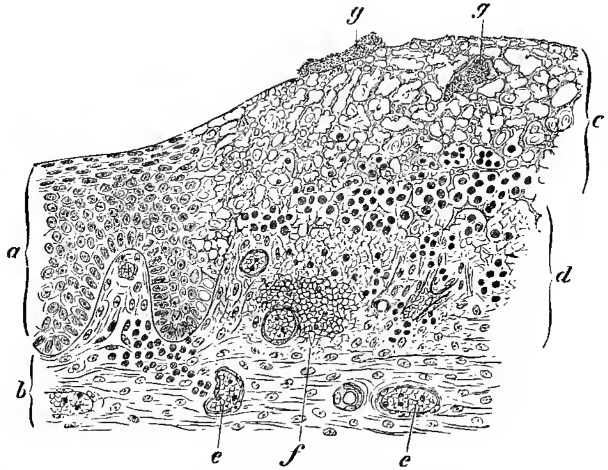

FIG. 312. SECTION THROUGH THE UVULA IN DIPHTHERIAL CROUPOUS PHARYNGITIS. (*Preparation hardened in alcohol, stained with Bismarck brown, and mounted in Canada balsam:* × 75)

a normal epithelium
b submucous connective tissue
c reticular fibrin enclosing a few epithelial cells and leucocytes
d submucous connective tissue infiltrated with coagulated fibrin and leucocytes
e blood-vessel
f haemorrhage
g clusters of micrococci

When the disease has lasted some days and the membranous deposits have reached a considerable thickness, they are frequently found to consist of a number of fibrinous layers. The most superficial layer, which is also the oldest, usually contains numerous

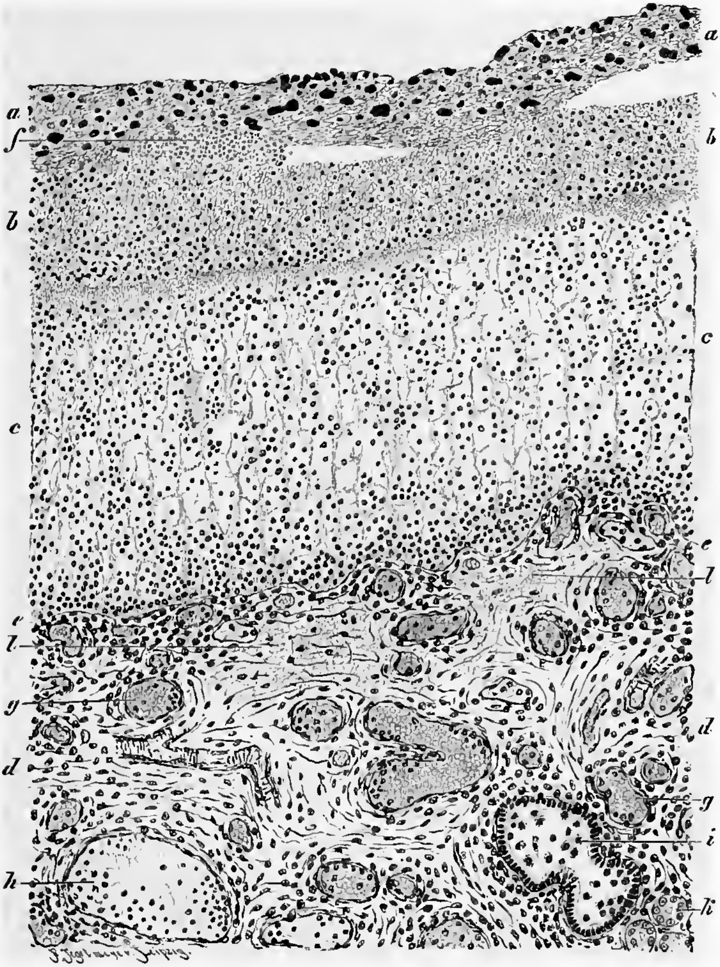

FIG. 313. DIPHTHERIAL CROUPOUS INFLAMMATION OF THE THROAT.

(*Section of an inflamed uvula covered with a laminated fibrinous membrane : preparation hardened in Müller's fluid, embedded in celloidin, stained with haematoxylin and eosin, and mounted in Canada balsam* : × 50)

a superficial fibrinous layer composed of epithelial scales and fibrin and beset with numerous colonies of micrococci
b second fibrinous layer consisting of a fine-meshed network enclosing leucocytes
c third fibrinous layer lying on the submucous connective tissue and composed of a coarse-meshed network enclosing leucocytes
d connective tissue infiltrated with cells
e infiltrated boundary-zone of the submucous connective tissue
f groups of red blood-corpuscles
g distended blood-vessels
h lymph-vessels distended with liquid, fibrin, and leucocytes
i duct of a mucous gland distended with secretion
k transverse section of gland-tubule
l fibrinous network in the superficial layers of the connective tissue

colonies of micrococci (Fig. 313 *a*) that have no causal relation to the disease. The other layers consist partly of fine-meshed fibrin (*b*), partly of a coarser network whose thicker strands often run at right angles to the surface (*c*). The meshes enclose nucleated leucocytes and liquid (*b c*) or liquid alone, or it may be pale hyaline flakes derived from dead leucocytes or epithelial cells. To judge from these structural appearances, the exudation and coagulation take place discontinuously, and in the main at places where the epithelium is degenerate and exfoliated, though the epithelium adjacent to these places may be partially overlaid and encroached on by the coagulated exudation. The fibrinous membranes formed in the earlier stages are usually closer and denser in texture (*b*) than the later deposits (*c*). The diphtherial bacilli of Löffler lie chiefly in the denser membranes (HEUBNER).

The tissue beneath the false membrane is always more or less infiltrated with cells (*d e*), and often also with fibrinous exudations (*l*) or even with blood. The blood-vessels are hyperaemic (*g*), the lymphatics dilated (*h*) and filled with fibrinous liquid, and the mouths of the mucous glands distended with mucus and desquamated epithelium (*i*). Very numerous fibrinous coagula are deposited in the swollen tonsils, and the lymphadenoid follicles are often pervaded by a dense network of fibrin (Fig. 314 *a*).

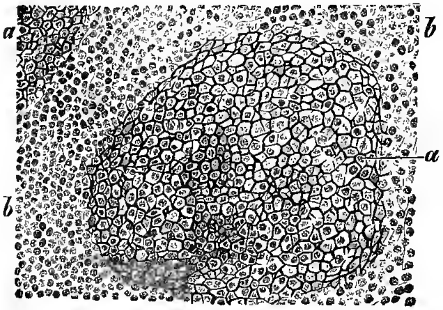

FIG. 314. FIBRINOUS NETWORK PERVADING THE LYMPHADENOID FOLLICLES OF THE TONSILS IN DIPHTHERIAL CROUPOUS PHARYNGITIS.

(*Preparation hardened in alcohol, stained with methyl-violet, and partially decolorised with iodine, xylol, and aniline oil:* × 150)

a follicle with fibrinous network
b intervening tissue containing lymphocytes

In most places the submucous connective tissue, though inflamed and infiltrated, survives, and therefore when recovery takes place by the separation of the false membrane, and the liquefaction and absorption of the infiltration, no cicatrix is formed. Some portions may however undergo necrosis, or in other words the inflammation of the submucous connective tissue becomes at certain points a **diphtheritic inflammation.** This result is commonest in the tonsils, whose lymphadenoid tissue is peculiarly liable to necrosis, and accordingly it not infrequently happens that portions of the tonsils are destroyed. Repair in such cases can take place only by the formation of a cicatrix.

The terms **diphtheritic** and **diphtherial** must be carefully distinguished. The former refers to *diphtheritis*, which is a mode of inflammation; the latter refers to *diphtheria*, which is a specific infective disease. *Diphtheritic* inflammation differs from catarrhal and croupous (or fibrinous) inflammation in that it implies necrosis and sloughing of part at least of the affected mucous membrane. The *diphtherial* virus, like the scarlatinal, may give rise to croupous as well as to diphtheritic inflammation. To *diphtherial* correspond the French word *diphthérique* and the German word *diphtherisch:* to *diphtheritic* correspond *diphthéritique* and *diphtheritisch.*

In **scarlatina** the inflammation extends over the mucous membrane of the mouth and pharynx, and is usually combined with marked swelling of the glands, and sometimes with diffuse inflammation of the cellular tissue of the neck. The mildest forms are of a catarrhal character, with intense reddening of the surface: in severer cases yellow or white spots, pellicles, and stouter membranes are formed on the fauces and palate, or on the tonsils and pharynx. These deposits, like the diphtherial membranes, are composed of coagulated fibrin, and generally enclose cast-off and necrotic epithelial cells. The submucous connective tissue, as in diphtheria, is infiltrated with cells and fibrin. In the severest forms the croupous inflammation passes into diphtheritic and gangrenous sloughing which involves the mucous membrane of the pharynx, fauces, and nose, and in some cases leads to very extensive destruction of tissue. The process is accompanied by the effusion of a foetid purulent secretion, and is liable to be followed by suppuration and gangrene of the lymph-glands and the cervical cellular tissue.

The ulcers of the throat that sometimes appear in **typhoid fever** are as a rule confined to the fauces (CAHN). They are flat circular and sharply-cut ulcers, with a diameter of from one to five millimetres, and are surrounded by a zone of hyperaemia: they heal up in a few days.

Phlegmonous inflammations and **abscesses** affect the pharynx and soft palate more frequently than the mouth. The swelling and redness at the outset are very intense. The purulent exudation accumulates mainly in the loose submucous tissue, and at length forms abscesses that break through to the surface. The commonest causes of suppurative inflammation of this kind are septic wounds and secondary infection from diphtheria or scarlet fever, or from glanders, syphilis, anthrax, and the like. Retropharyngeal abscesses are sometimes produced in connexion with caries of the cervical vertebrae. Such abscesses endanger life by causing erosion of blood-vessels, or by producing obstruction of the glottis through excessive oedema of the surrounding mucous membrane. General septicaemia and pyaemia are occasionally induced by phlegmonous inflammation of the throat.

Syphilitic affections of the palate and pharynx have already been described in Art. 175. **Tuberculosis** gives rise to subepithelial granulations containing tubercles on the palate, fauces, tonsils, and pharyngeal walls. As these break down they leave ulcers, and in some cases the greater part of the pharyngeal mucous membrane is thus destroyed. The tonsils and follicular glands are very susceptible to tuberculous infection, and in phthisical patients are frequently found to be morbidly altered thereby.

Tumours of the throat are rare, but examples of histioid, epithelial, and teratomatous growths have been observed (Art. 178).

Thrush appears in the throat under the same conditions as in the mouth.

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CHAPTER LXI

THE SALIVARY GLANDS

184. The salivary organs are racemose glands whose secretion is discharged into the oral cavity. The chief disorders to which they are liable are those due to inflammations and to the growth of tumours.

Mumps or epidemic parotitis is a specific infective inflammatory swelling of the parotid gland. The submaxillary and sublingual glands are occasionally affected at the same time. The glands and the overlying tissues are much enlarged, and feel doughy to the touch.

Similar glandular swellings occur as a secondary symptom in connexion with certain infective disorders like typhoid, cholera, pyaemia, syphilis, diphtheria, etc.

The swelling is due to an inflammatory serous and cellular infiltration of the connective tissue in and around the acini. It ends either in resolution, in fibroid induration, or in suppuration and abscess. Sometimes gangrene supervenes. The micro-organisms which induce the inflammation probably gain access to the gland from the mouth by way of the glandular ducts. In purulent inflammations at any rate the pyogenic micrococci often lie mainly in the ducts. Mumps is liable to be followed by what appear to be metastatic inflammations of the testes, ovary, bladder, and kidneys. In some cases multiple arthritis or synovitis has been recorded as a sequela.

Angina Ludovici or sublingual **cynanche** is an acute phlegmonous inflammation of the tissue surrounding the submaxillary gland, resulting in suppuration or gangrene.

Milder forms of inflammation, both acute and chronic, are also met with in connexion with disorder of the salivary glands, resulting from mechanical injury or retention of their secretion or from other causes not easy to determine. When chronic they lead to fibrous hyperplasia, while the gland-substance often becomes atrophied. If the duct becomes involved in a contracting cicatrix it may be obstructed or altogether occluded.

Syphilitic inflammation of the salivary glands is rare, though cases of gummatous infiltration have been described by LANCEREAUX, FOURNIER, VERNEUIL, LANG, and others: it leads to disintegration or to cicatricial induration of the affected tissue.

A **salivary fistula** is an opening or channel connecting a salivary duct with the surface of the buccal mucous membrane on the one hand or the external skin on the other. It results either from a wound or from some suppurative inflammation leading to perforation.

When a salivary duct is obstructed or occluded at any point the smaller ducts behind the obstruction become dilated by the retained secretion. These dilated ducts are uniformly cylindrical, fusiform, or even pear-shaped. As the accumulation goes on the ducts and the lumen of the gland become distended into globular **cysts**, often of very considerable size.

The cysts produced by dilatation of the submaxillary and sublingual ducts protrude from beneath the tongue, and are often spoken of as **ranulae**, like those which arise from dilatation of the mucous glands of the tip of the tongue (Art. 179).

Salivary calculi are stony concretions which form occasionally in Stenson's and Wharton's ducts. They consist of calcium phosphate and carbonate. Sometimes they enclose foreign matters which have accidentally gained access to the ducts.

Tumours both of the connective-tissue and of the epithelial type are met with in the salivary glands. Of the former class chondroma, myxoma, fibroma, sarcoma, and rhabdomyoma may be mentioned. They usually give rise to sharply-defined nodes or nodules. Carcinoma generally begins as an isolated nodule, which extends so as to involve the whole gland, and then invades the surrounding tissues.

These neoplasms are very apt to exhibit a mixed type of structure, especially those of the parotid gland. Thus cartilaginous, myxomatous, sarcomatous, and fibromatous elements may all occur within the same tumour. Sometimes the peculiar hyaline formations characteristic of cylindroma are met with. Combinations of carcinoma with sarcoma or chondroma have been observed.

Xerostomia or dry mouth is a condition in which the salivary secretion is abnormally diminished or suppressed. The tongue and buccal mucous membrane are raw, fissured, and dry; but there is no apparent alteration in the ducts or acini of the salivary glands. The condition is attributed to some disorder of the nervous secretory mechanism.

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CHAPTER LXII

THE OESOPHAGUS

185. Among the various congenital **deformities** to which the oesophagus is liable in otherwise well-formed infants there is one in which the anatomical appearance of the part is nearly always the same. In this form the oesophagus ends in a blind pouch in its upper third (Fig. 315 *c*), and after a short interruption re-appears as a tube (*e*) communicating at its upper end with the air-passage by an opening (*d*) in the posterior wall of the trachea. The ends of the two segments are either entirely separate or connected only by a solid cord. The level of the opening into the trachea varies in different cases: in rare instances the air-tube opens into the upper oesophageal segment.

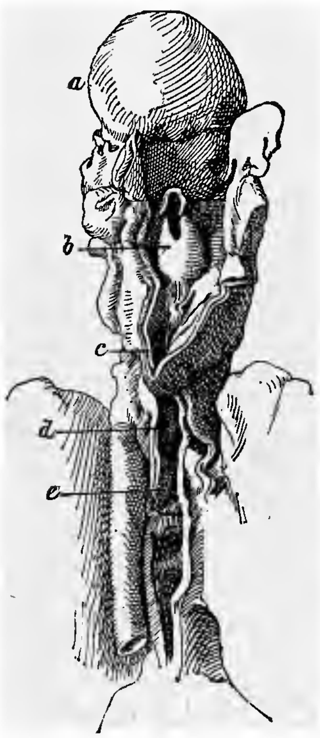

FIG. 315. MALFORMATION OF THE OESOPHAGUS.

(*Reduced to two-thirds of the natural size*)

a tongue *b* larynx
c blind end of the oesophagus
d opening in the posterior wall of the trachea
e lower segment of the oesophagus

Other rare malformations, unaccompanied by further developmental anomalies, are oesophago-tracheal fistulae and occlusion or stenosis of the oesophagus. Stenoses may exist either in the upper or in the lower portion of the tube, and are simply annular or extend over some small distance. Lateral diverticula and fistulous passages occur at different levels in the pharyngeal portion, due to persistence of the foetal branchial pouches; these are accordingly to be regarded as internal branchial fistulae, incomplete or complete as the case may be.

The most important of the acquired deformities to which the oesophagus is liable is **stenosis** or constriction of the tube, due to compression, obstruction, stricture, or spastic muscular contraction.

Stenosis by **compression** is generally due to enlargement of the thyroid gland, as in goitre, to tumours of the lymph-glands of the neck or mediastinum, to mediastinal sarcoma, to aneurysm of the aorta, etc. It gives rise to grave functional disturbance only when the tube is in a manner encircled by the growth, so that there is no direction in which it can yield.

Stenosis by **obstruction** occurs when foreign bodies become impacted in the oesophagus. The thrush-fungus sometimes appears in the oesophagus and grows to such an extent as occasionally to block up or seriously to narrow the passage. Polypous tumours growing from the mucous membrane may have a like effect, but very rarely. Cancerous growths are much more apt to cause obstruction.

Strictures are due to the contraction of cicatrices or to cancerous infiltration. Cicatrices most commonly follow upon injuries due to irritant or corrosive substances, such as boiling water, acids, or alkalies. If the corrosion has extended deeply into the tissues of the wall, the oesophagus is liable to be transformed into a firm cicatricial tube, through which only the finest sound may be able to pass. Syphilitic strictures are very rare, as the oesophagus is on the whole seldom attacked by syphilitic inflammation. Cancerous strictures are due to infiltration of the whole circumference of the oesophagus by the neoplasm, by which it is transformed into a kind of tough unyielding tube (Fig. 321 *b*), the infiltrated tissue often at the same time undergoing contraction: they extend over a length of 5 to 10 centimetres, and the inner surface is generally ulcerated.

Simple **dilatation** of the oesophagus is generally the result of stenosis of its lower portion (Fig. 321 *a*) or of the cardiac orifice of the stomach. In this case the muscular walls yield and become distended as the ingested food gathers above the contracted portion. The dilatation is generally uniform, but occasionally it is unilateral, and in this way diverticula are sometimes produced. The various coats are often thickened in the dilated portion.

But dilatation may take place without stenosis (Fig. 316), and in this case the oesophagus assumes the form of a fusiform sac, the wall of which, especially the muscular coat and at times the mucous membrane also, is more or less thickened, and not infrequently beset with small ulcers. Whether the condition is due to temporary spastic contraction of the cardiac orifice of the stomach, or to abnormal weakness of the muscular coat, is difficult to determine: the thickness of the muscular layers would however seem to indicate the former alternative. It is of course possible that the condition is dependent on some developmental anomaly. Local dilatations above the diaphragm are said to occur as congenital malformations (LUSCHKA, ZENKER).

Diverticula appear as local sacculations at some part of the wall of the oesophagus or pharynx. They are due to pressure from within or traction from without (ZENKER).

Diverticula of the former class are due to pressure from within tending to push out the wall. They are formed usually in the pharyngeal region, and according to their situation are distin-

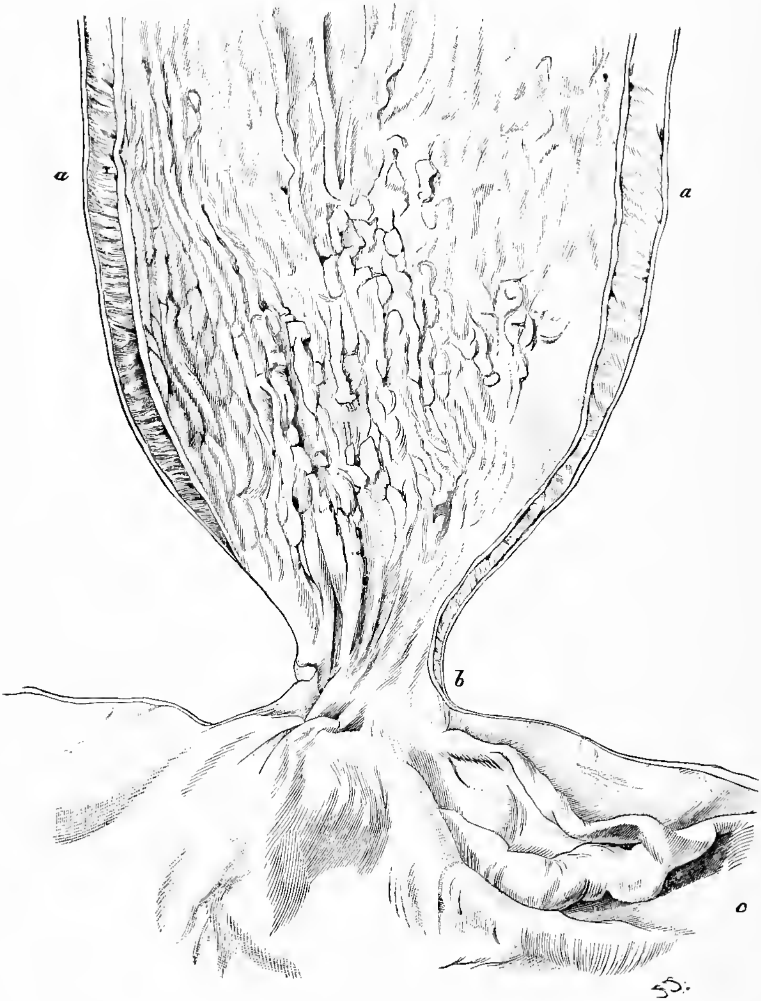

FIG. 316. DILATATION AND HYPERTROPHY OF THE OESOPHAGUS.

(*The condition had existed for at least twelve years before death: four-fifths of the natural size*)

a oesophagus, circumference enlarged to 10 centimetres, with thickened walls and corrugated mucosa beset with numerous small ulcers
b cardiac orifice
c stomach

guished as dorsal or lateral. Dorsal diverticula occur at the lower end of the pharynx, and appear either as small sharply-defined pouches of the size of a hazel-nut or less, shallow or deep, and directed posteriorly, or as large globular, cylindrical, or pyriform sacs (Fig. 317 *d*) hanging down between the oesophageal tube and the spine. The walls of such a sac are moderately thick, and consist of thickened mucosa and submucosa with external adventitious layers of fibrous tissue: the muscular coat is either entirely absent or persists only around the neck of the sac. The diverticulum is in fact a hernia (pharyngocele) of the mucous membrane through the muscular bundles of the inferior constrictor of the pharynx. It is due to some local weakening of the posterior wall of the pharynx, permitting the inner layers to be pushed through the outer under the internal pressure exerted in the act of swallowing. The weakening is often induced by some mechanical injury to the pharyngeal wall, such as that caused by the impaction of a foreign body. As food sometimes lodges in the diverticulum and remains there decomposing for a time (Fig. 317 *d*), it is liable to act as an irritant to the mucous membrane and give rise to inflammatory thickening of the wall, or occasionally to mucous papillary growths from its inner surface.

Lateral diverticula, when they are not simply persistent branchial pouches, are due to the secondary dilatation of incomplete branchial fistulae.

Diverticula due to traction from without (Fig. 318 *b*) are in general situated on the anterior aspect of the oesophagus, and most commonly at the level of the bifurcation of the trachea. They are usually narrow and funnel-shaped (*b*), varying in depth from 2 to 20 millimetres, with the apex pointing directly forwards or a little to one side. Simple shallow bulgings are more rare. The funnel consists of mucosa and submucosa, which may be wholly or partially or not at all covered with a muscular layer. The apex almost invariably runs out into a band of dense fibrous tissue, generally con-

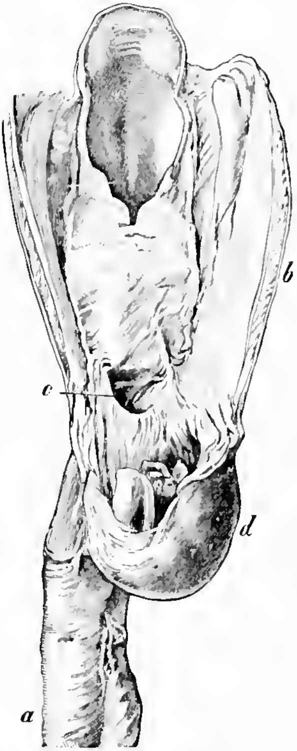

FIG. 317. PRESSURE-DIVERTICULUM OF THE PHARYNX AT ITS JUNCTION WITH THE OESOPHAGUS.

(*Seen from behind, and partially opened: two-thirds of the natural size*)

a oesophagus *b* pharynx
c opening into the oesophagus
d diverticulum containing remnants of food (plum-stone)

taining shrunken bronchial glands (*c*) and connected with the trachea or one of the bronchi. The diverticulum thus appears to be ultimately due to an inflammatory process starting in some lymph-gland and involving by extension the wall of the oesophagus. It is the contraction of the resulting inflammatory or cicatricial tissue that gives rise to external traction upon the oesophageal wall. The diverticulum has no tendency to enlarge, but it may be perforated, especially when a foreign body becomes wedged in it.

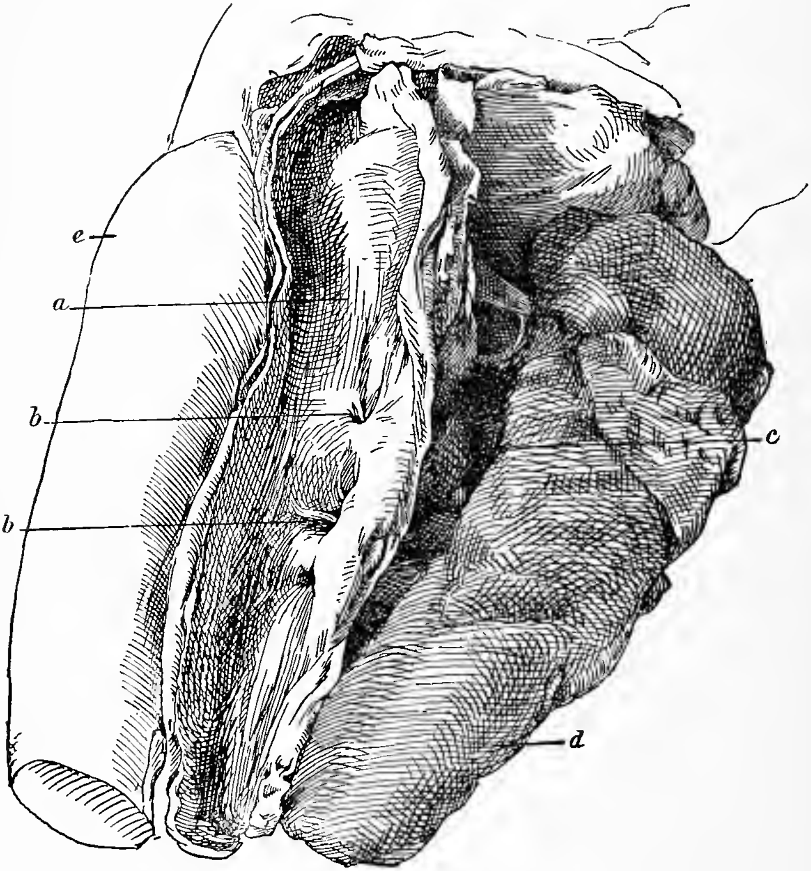

FIG. 318. TRACTION-DIVERTICULA OF THE OESOPHAGUS.
(*Five-sixths of the natural size*)

a oesophagus
b diverticula
c cluster of hardened and shrunken lymph-glands
d lung
e descending aorta

Rupture of the healthy oesophagus is rare, if we leave out of account the cases in which it is directly wounded from without. There are however some instances on record in which strangulation or violent vomiting has led to longitudinal or transverse rents of the wall. It may be that in these latter cases the tissues were to some extent softened by the regurgitated gastric juice (*oesophagomalacia*). This digestive softening is not uncommon as a post-mortem phenomenon: the affected tissue appears grey or yellow and sodden and it is readily torn. According to ZENKER it is apt to occur *in articulo mortis;* but the instances must be very rare in which it takes place in a healthy patient. According to QUINCKE, CHIARI, and KEHRER, **round ulcers** of the oesophagus (*ulcus ex digestione*), analogous to the round or perforating ulcer of the stomach, are occasionally met with.

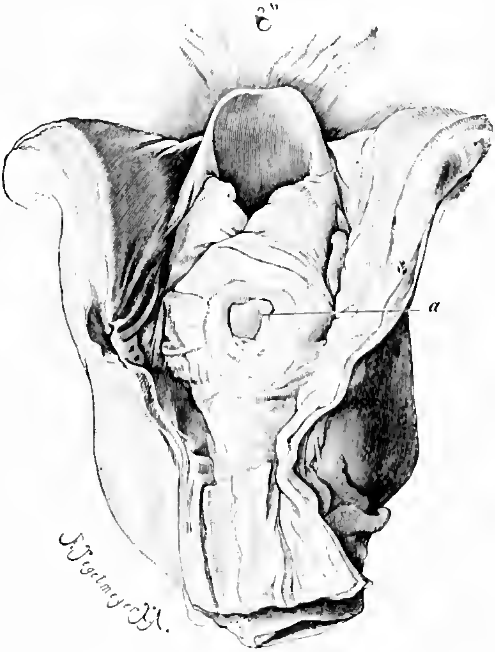

FIG. 319. DECUBITAL NECROSIS OF THE PHARYNGEAL WALL.

(*Five-sixths of the natural size*)

a perforation of the pharyngeal wall through which a portion of the exposed cricoid cartilage is visible

Perforation of the oesophagus and pharynx is due to disease in the tube itself or in the adjoining parts. Cancerous ulceration (Fig. 320) and the impaction of foreign bodies are the commonest causes of the former; corrosion by ingested liquids and simple ulceration come next in point of frequency. In the pharyngeal region gangrenous necrosis leading to perforation is sometimes met with in the anterior and posterior wall of the portion lying behind the cricoid cartilage (Fig. 319 *a*). It occurs in debilitated and bed-ridden patients, and is due to continuous compression of the tube between the larynx and the spine, the extreme relaxation of the muscles permitting the larynx to bear down on the yielding pharynx: it is therefore of the same nature as a bed-sore or decubital necrosis. Perforation of the oesophagus from without may be due to caseous and suppurating lymph-glands, vertebral abscesses, gangrene of a goitrous tumour, or aneurysm of the thoracic aorta.

Perforation is always followed by more or less extensive inflammation of the neighbouring parts, which is often purulent or gangrenous in character.

Varicose dilatation of the oesophageal veins sometimes takes place, chiefly in cases of cirrhosis of the liver; it is liable to give rise to serious haemorrhage.

186. **Catarrhal inflammation** of the oesophagus is marked chiefly by epithelial desquamation; but little mucus is poured out in the chronic forms, and in the acute forms it is absent. The desquamated cells give the mucous surface a dull whitish or yellowish tint. Sometimes minute superficial ulcerations occur. When the inflammation is caused by the presence of a foreign body, a deep ulcer is often formed at the spot where the latter is in contact with the wall.

In chronic catarrh the mucous membrane may become hypertrophied, and papillomatous or polypous outgrowths are apt to arise from its surface: the muscular coat may also show signs of hypertrophy. If the mucous glands become obstructed they give rise to little cysts in the mucous membrane, which are apt to suppurate (CHIARI) and become converted into minute abscesses and ulcers.

Croupous and **diphtheritic inflammation** are rare. They appear most frequently in connexion with typhoid, cholera, measles, scarlatina, small-pox, pulmonary tuberculosis, and pyaemia: they very seldom indeed accompany diphtheria. Sometimes in the course of small-pox regular variolous pustules are formed in the oesophagus.

Phlegmonous inflammation is either local or diffused over a considerable area: it is however an extremely rare affection. If the collection of pus which forms in the submucous tissue breaks through the surface, complete repair and recovery may ensue. When the phlegmonous abscess is larger, undermining a considerable portion of the mucous layer and breaking through it at several points, some part of the cavity of the abscess may persist after its evacuation: it becomes gradually covered over with epithelium growing in from the sites of perforation. This variety of inflammation is due to wounds or corrosions, or to the extension upwards of a phlegmonous inflammation of the stomach, or to purulent inflammation extending inwards from the surrounding tissues.

Corrosive substances like sulphuric, nitric, or hydrochloric acids, caustic potash or soda, copper sulphate, etc., give rise to more or less wide-spread destruction of the oesophageal tissues. If the acids are dilute the epithelium alone may be destroyed, becoming white and turbid and falling away from the mucosa, sometimes in large shreds. When the corrosive action goes further, the mucous membrane in its whole thickness is transformed into a grey or brown or black slough, and sometimes the muscular coat is destroyed likewise. If the patient survives, wide-spread inflammation results, which is usually suppurative and now and then leads to perforation. When however the suppuration causes the necrosed tissue to separate, the wound becomes scarred over; and if the muscular coat has been destroyed the scar contracts and gives rise to extreme constriction of the tube.

Syphilitic and **tuberculous inflammation** and ulceration are extremely rare in the oesophagus. Ulceration commonly takes place when tuberculous lymph-glands break through into the oesophageal wall.

Connective-tissue tumours of the oesophagus are not common, though fibroma, lipoma, myxoma, and sarcoma are sometimes met with. As a rule, they form globular polypoid growths. This is especially true of fibroma, which occasionally developes in the lower part of the pharynx behind the larynx, and hangs pendulous within the tube.

Papillomatous outgrowths from the mucous membrane are more common than true connective-tissue tumours. They somewhat resemble condylomatous warts in their general structure.

Carcinoma is however by far the most important of the neoplasms affecting the oesophagus. It may appear at any point of the tube, though it is most frequently met with in the lower third. It is usually of the squamous epithelial type, and forms isolated patches, or encircling growths, which soon become ulcerous (Fig. 320). Sometimes the protuberant parts of the growth are entirely removed by ulceration, while the base and margins of the sore continue to be infiltrated with cancer-tissue. Certain parts of the growth continue to ulcerate, while others become indurated with shrunken cicatricial tissue, and so give rise to extreme stenosis (Fig. 321 *b*). The infiltration and fibrous hyperplasia extend to the muscular coat, and then to the

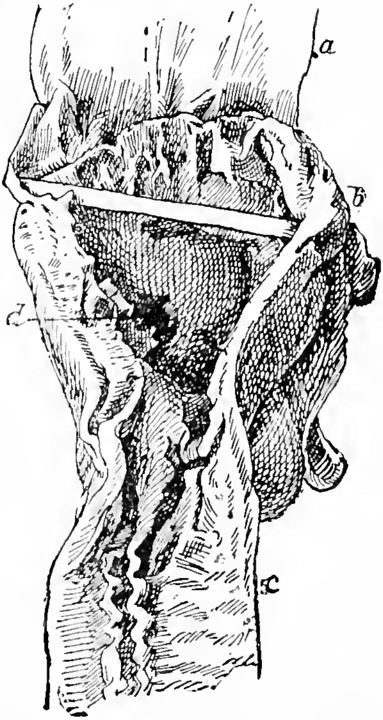

FIG. 320. CARCINOMATOUS ULCER OF THE OESOPHAGUS PENETRATING INTO THE LARYNX.

(*Five-sixths of the natural size*)

a oesophagus above the ulcer
b ulcer
c oesophagus beneath the ulcer
d point of perforation

adjacent tissues and organs. Later on the trachea, bronchi, pericardium, heart, pleura, and lungs may be successively invaded by the cancerous infiltration. If the oesophagus is perforated (Fig. 320 *d*) by deep ulceration, the process of ulcerous disintegration rapidly extends thence into the contiguous parts, the tissue of which is always more or less inflamed.

Thrush sometimes appears in the oesophagus as well as in the mouth and throat (Art. 176).

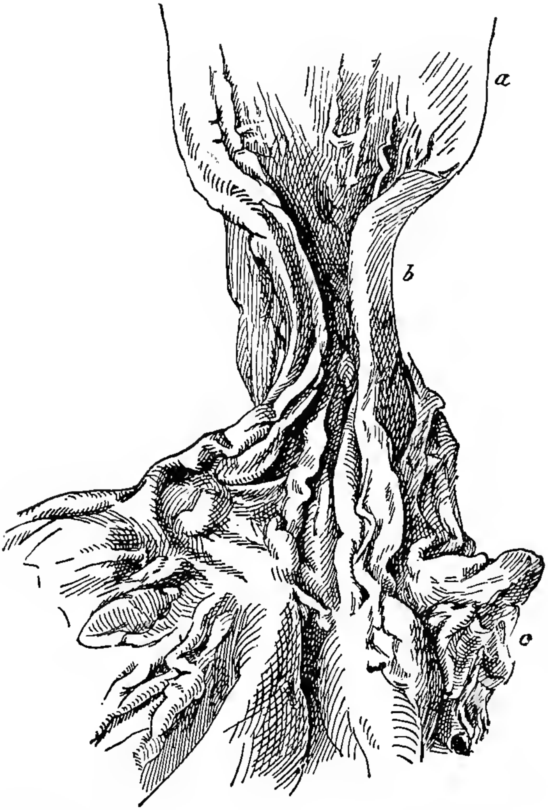

FIG. 321. CARCINOMATOUS STRICTURE OF THE OESOPHAGUS ABOVE THE CARDIAC ORIFICE.

(*Five-sixths of the natural size*)

a dilated oesophagus *b* constricted, indurated, thickened, and ulcerated portion of the oesophagus *c* stomach

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CHAPTER LXIII

THE STOMACH

187. **Congenital anomalies.** The stomach is occasionally absent in acephalous monsters. In rare cases it is found to be abnormally small in foetuses that are otherwise well developed. Complete atresia of the pylorus is very rare, but stenosis or abnormal contraction is not infrequent (R. Maier).

Of congenital anomalies of form we may mention abnormal constrictions of the stomach giving it an hour-glass shape, and the occurrence of partitions abnormally subdividing it. In cases of *Situs transversus*, or lateral transposition of the viscera, in persistent fissure of the abdominal wall, and in congenital deficiency of the diaphragm, the stomach is as a rule misplaced. Sometimes the vertical position of foetal life persists in the adult.

Among **acquired anomalies** of form and position, dilatation of the stomach is the commonest, and is due as a rule to abnormal narrowing of the pylorus. In other cases it occurs as a result of abnormal position or morbid adhesions of the organ, of distension from excess of ingesta, or from textural changes in the walls.

In extreme dilatation the stomach occupies a large extent of the abdominal cavity, extending backwards rather than forwards. It may reach from the left side of the diaphragm to the symphysis pubis, compressing the bladder and covering over almost the whole of the intestinal convolutions. The left half of the lesser curvature runs parallel to the spine, in continuation of the line of the oesophagus; the pyloric half bends up at a sharp angle towards the liver. The greater curvature lies along the left side of the abdomen to the point of flexure in the pyloric portion, the pylorus being dragged more or less backwards and the hepatico-duodenal ligament being stretched. The coats of the stomach may be thinned in every part or, according to the cause of the dilatation, they may be thickened at various places, especially in the neighbourhood of the pyloric end.

Acquired contraction or constriction of the stomach is due either to functional inactivity of the organ, as in prolonged starvation, or to inflammation, ulceration, or new-growth, leading to cicatricial contraction. Peritonitis, followed by adhesions and contraction of the serous membrane (*peritonitis deformans*), may give rise to shrinking or other deformation of the organ.

Pyloric stenosis is in general due to the cicatrisation of gastric ulcers or to new-growths in the mucous membrane; it is also apt to follow all processes, like carcinomatous ulceration, that give rise to induration and thickening of the stomach-walls.

Partial alterations of form are due to local disease. Ulcers (chiefly along the lesser curvature) that heal and become cicatrised lead to contractions and constrictions, which may be so extreme as to give the stomach the form of an hour-glass, or to bring the cardiac and pyloric ends almost into apposition. Gastric diverticula are very rare.

Displacements of the stomach as a whole may be caused either by changes in the surrounding parts or by disorders of the organ itself.

Notable **thickening of the walls** of the stomach is most frequently caused by carcinomatous disease (Art. 193), and chiefly affects the pyloric end. The thickening in such cases may be due not only to cancerous infiltration, but to the formation of new connective tissue, and in some degree to hypertrophy of the muscular coat. Simple inflammatory processes lead but rarely to any considerable thickening of the gastric wall (Art. 189).

Hypertrophy of the muscular coat of the stomach developes most frequently as a result of stenosis of the pyloric orifice. It may however take place in consequence of cancerous and inflammatory disease, or of functional disturbances unaccompanied by any demonstrable narrowing of the pylorus. Muscular hypertrophy is generally most marked near the pylorus, whose thickness is in some instances remarkably increased.

Rupture of the stomach is not infrequently the result of ulcerous destruction of the gastric wall, or of traumatic injury. Ruptures from rapid over-distension of the stomach, unaccompanied by antecedent ulceration, are very rare. The point of rupture is usually in the lesser curvature.

Atrophy of the coats of the stomach is met with in conditions of general cachexia, and in cases of dilatation. In these and in other conditions fatty degeneration of the muscular fibres has been observed. When the fibrous elements become hyperplastic the muscular tissue is not infrequently atrophied, while the glandular structures of the mucosa dwindle and degenerate (Art. 189).

References on Malformations and Acquired Deformities of the Stomach.

DEMME: Dilatation of the stomach in children *Jahresber. d. Kinderspitals* Berne **1882**
EWALD: *Die Krankheiten des Magens* Berlin **1893** (trans. by SAUNDBY) London **1891**
KUSSMAUL: *D. A. f. klin. Med.* vi **1869**, and *A. gén. de méd.* xv **1870**
LANDERER: *Die angeborene Stenose des Pylorus* Tubingen **1879**
LESSHAFT: The position of the stomach *V. A.* 87 **1882**

LEUBE: *Ziemssen's Cyclop.* VII New York **1876**, and *D. A. f. klin. Med.* XXIII **1879** and XXXIII **1883**
MACHON: *La dilatation de l'estomac chez les enfants* Geneva **1887**
MAIER, R.: Congenital stenosis of the pylorus *V. A.* 102 **1885**
NAUWERCK: Hypertrophic stenosis of the pylorus *D. A. f. klin. Med.* XXI **1878**
PENZOLDT: *Die Magenerweiterung* Erlangen **1875**
PERTIK: Aetiology of dilatation of the stomach *V. A.* 114 **1888**
POENSGEN: *Die motorischen Verrichtungen des menschlichen Magens* Strassburg **1882**
STRUTHERS: Cases of double stomach *Anat. and physiol. observations* Edinburgh **1854**
THIÉBAUT: Dilatation of the stomach *Thèse* Nancy **1882**
TILGER: Stenosing pyloric hypertrophy *V. A.* 132 **1893**; Congenital stenosis *ibidem* 133 **1893**
VON WUNSCHHEIM: Spontaneous rupture of the stomach *Prag. med. Woch.* **1893** (with references)

188. After food or drink is swallowed, the **ingesta** linger in the stomach for a time, and are in part directly absorbed by the gastric mucous membrane; the remainder, either altered by gastric digestion or unaltered, is passed on through the pylorus into the small intestine.

The ingesta always act as a stimulus to the gastric walls, and in particular to the mucous membrane. The stimulus calls into play the functional activity of the stomach, the muscular coat contracting, and the mucous membrane secreting gastric juice by means of its glands, together with a certain amount of mucus from its superficial epithelium.

Among the substances that occasionally reach the stomach, there are many which by reason of their chemical and physical properties stimulate or irritate the stomach-wall in a pathological manner; and even ordinary foods may act injuriously when they are taken too hot or too cold. Of such injurious substances the most important are the corrosive poisons, and those that have the property of rapidly abstracting water from the tissues.

When ordinary food is introduced into the stomach in excessive amount, so that the stomach cannot deal with it by normal digestion and after a time pass it on into the small intestine, the gastric mucous membrane is liable to be injured. The risk of injury is increased when the stomach, from any cause such as narrowing of the pylorus, weakness of the muscular coat, or fibroid induration of the wall, is incapable of completely emptying itself, and when its contents include fermentive micro-organisms such as those that produce lactic acid, butyric acid, acetic acid, or putrid decomposition. The products of these fermentations act injuriously upon the mucous membrane, and disturb or inhibit the functions of the stomach.

The pathological effects induced by noxious ingesta take the form either of abnormal stimulation or of weakening and inhibition of the gastric functions. They may also give rise to demonstrable anatomical changes, such as hyperaemia, increased production of mucus, degeneration of the superficial epithelium and gland-cells, inflammation, or even necrosis of the tissues.

The most marked changes are those induced by the action of **corrosive poisons,** which, according as they are more or less concentrated, give rise to necrosis and sloughing, or to degeneration and inflammation. The damage often extends to the mouth, pharynx, and oesophagus, and even to the small intestine, the mucosa of the latter being very readily affected by such poisons, even when much diluted.

All corrosive poisons, whether acids or alkalies, when concentrated give rise to sloughing and separation of the superficial layers of the mucous membrane (A. LESSER). The sloughs or eschars caused by sulphuric acid are greyish-white, dry and coarse in appearance, and brittle. When recent the several elements of the tissue are still recognisable in the slough, though they are turbid and shrunken. The sloughs due to hydrochloric acid are similar. Nitric acid produces a yellow or orange slough. The colour of the less-affected parts is pale purple or greyish. Oxalic acid gives rise to slight and superficial sloughs, which are white or greyish in colour. A concentrated solution of caustic potash acts like sulphuric acid, but the sloughs are less brittle. Parts that have been for some time in contact with the alkali become semi-transparent. Corrosive sublimate, carbolic acid, and arsenious acid give rise to white sloughs.

The mineral acids and alkalies are the most powerfully corrosive. Not only may all the coats of the stomach be destroyed by their action, but the neighbouring organs may be similarly corroded and discoloured. The liver and spleen are especially liable to be thus attacked, and then look almost as if they had been boiled.

In the parts surrounding the sloughs, and in other places where the poison has been somewhat diluted, a more or less intense and often haemorrhagic inflammation is set up. The parts thus affected presently take on a brownish, greenish, or greyish-black tint. The slough at the same time softens or breaks down, and this more readily in the case of acids than of alkalies. By and by the dead tissue is cast off and liquefied.

Intense inflammation is set up by the strong mineral acids; oxalic acid, corrosive sublimate, carbolic acid, and arsenious acid are much less active in this respect. It should however be remarked that arsenic often gives rise to an abundant secretion of mucus; and the tenacious slough-like shreds that in recent cases cover the inner surface of the stomach, especially on the ridges of the corrugations, consist in part simply of masses of coagulated mucus. According to KAUFMANN, in cases of poisoning by corrosive sublimate, calcareous granules are sometimes deposited in the epithelial cells, and appear as yellowish-white spots.

If the patient does not straightway die, the corrosion-wounds may heal by cicatrisation. In parts where the action of the corrosive agent has been most intense, the glandular layer may be totally destroyed, and the *muscularis mucosae* and the submucosa indurated. When the inner layer of the mucosa is alone destroyed, the indurated scar-like tissue contains remnants of glands, some of which degenerate into small cysts, and with these the inner surface of the cicatrix is often thickly beset. Where the corrosion has been extensive, very great contraction of the stomach and intestine may result.

Haematogenous degeneration of the gastric mucous membrane occurs chiefly in the course of infective and toxic affections, such as small-pox, septicaemia, typhoid fever, ulcerative pulmonary tuberculosis, phosphorus-poisoning, chronic lead-poisoning, and nephritis. It generally takes the form of cloudy swelling, fatty degeneration, and desquamation of the glandular epithelium, which give a turbid whitish appearance to the gastric mucous membrane. Sometimes shrinking, dropsical swelling, and vacuolation of the epithelium are observed. These changes may be combined with inflammatory infiltration and fibrous hyperplasia, especially in the gastric affections accompanying pulmonary phthisis (MARFAN, SCHWALBE, STINTZING), due in part at least to the swallowing of tuberculous sputum.

In amyloid degeneration of other organs the fibrous constituents of the vessels of the gastric mucous membrane are sometimes the seat of amyloid deposits.

References on the Action of Corrosive and other Poisons upon the Alimentary Tract.

BÖHM: *Ziemssen's Cyclop.* XVII New York **1879**

CASPAR-LIMAN: *Handb. der gerichtlichen Medicin* Berlin **1889**

EBSTEIN: Changes in the gastric mucous membrane from alcohol and phosphorus *V. A.* 55 **1872**

FILEHNE: Changes in the stomach in arsenical poisoning *V. A.* 83 **1881**

IPSEN: Poisoning by nitric acid *Vierteljahrsschr. f. gerichtl. Med.* VI **1893**

KAUFMANN: *Die Sublimatvergiftung* Breslau **1888**, and *V. A.* 117 **1889**

LESSER: Changes in the alimentary tract due to corrosive poisons *V. A.* 83 **1881**

LETULLE and VAQUEZ: Poisoning by hydrochloric acid *A. de physiol.* I **1889**

LEWY, B.: Morbid anatomy of the stomach (poisoning by nitric acid) *Ziegler's Beiträge* 1 **1886**

MAIER, R.: Lead-poisoning *V. A.* 90 **1882**

SACHS: The gastric mucous membrane in disease *A. f. exp. Path.* XXII **1887**

VIRCHOW: The condition of the stomach in phosphorus-poisoning *V. A.* 31 **1864**

References on Secondary Affections of the Stomach in Infective and other Diseases.

EDINGER: The gland-cells of the stomach *A. f. mikrosk. Anatomie* XVII **1879**

FENWICK, W.: The relation of certain morbid conditions of the stomach to affections of other organs *V. A.* 118 **1889**

KUPFFER: *Epithel und Drüsen des menschlichen Magens* Munich **1883**
MARFAN: *Troubles et lésions gastriques dans la phthisie pulmonaire* Paris **1887**
SACHS: The gastric mucous membrane in disease *A. f. exp. Path.* XXIV **1887**
SCHWALBE: The gastritis of phthisis *V. A.* 117 **1889**
SMIRNOW: Membranous and diphtheritic gastritis *V. A.* 113 **1886**
STINTZING: The structure of the diseased gastric mucous membrane *Münch. med. Woch.* XXXVI **1889**

189. **Inflammation** of the stomach is usually caused by changes in its contents (Art. 188); but it is sometimes of haematogenous origin, and in other instances is due to the extension of inflammation from the surrounding parts. In recent acute catarrh (catarrhal gastritis) the mucous membrane of the stomach appears dark-red and swollen, and is occasionally beset with small haemorrhagic patches. The surface is covered with a semi-transparent viscid film consisting of mucus, mucoid epithelium, and leucocytes. The cylindrical epithelial cells of the gland-ducts, which in normal conditions produce large quantities of mucus from their protoplasm, are seen to have passed into an extreme stage of mucoid change, and many are in process of desquamation. The epithelial cells of the peptic glands lie loosely in the lumen, and seem more granular than usual. The vessels of the interglandular connective tissue are distended, and their course is marked by cellular infiltration of the surrounding tissue, especially along the small veins. The subglandular tissue, and in some cases the submucosa, are here and there infiltrated; the endothelium of the lymphatics is swollen and desquamating, and some of the cells are multinuclear. These signs of inflammation may appear over the whole extent of the mucous membrane, or may be confined to a few patches; very often the pyloric end alone is affected.

Croupous and **diphtheritic inflammations** of the mucous membrane, other than those due to corrosive poisoning (Art. 188), are rare. They occur most frequently in connexion with diphtheria, scarlatina, and small-pox, and in infants who die of septic inflammation about the umbilicus. The croupous exudation takes the form of more or less extensive greyish-yellow false membranes lying on the reddened epithelial surface; but these very rarely extend over any large part of the gastric wall. In diphtheritic inflammation the necrotic membrane may consist only of the superficial epithelial cells (and then appears as a small greyish patch); or it may include the whole thickness of the mucosa, which is then transformed into a grey or blackish slough.

Most of the inflammations of the stomach are transient affections terminating in resolution and recovery; but they may pass into a chronic form, and so lead to permanent changes. Thus the inflammatory infiltration and the epithelial desquamation and destruction may become extreme. When the process lasts for some time and the regeneration of the epithelium is incomplete, partial atrophy of the glandular structures is liable to take place. Suppuration and diphtheritic sloughing lead to destruction of the connective tissue; extensive haemorrhagic infiltrations are followed by tissue-necrosis and liquefaction, accelerated by the dissolving action of the gastric juice (Art. 191). In this way more or less extensive loss of substance takes place in the mucosa, giving rise to superficial or deep **ulcers**, whose size depends on their mode of origin.

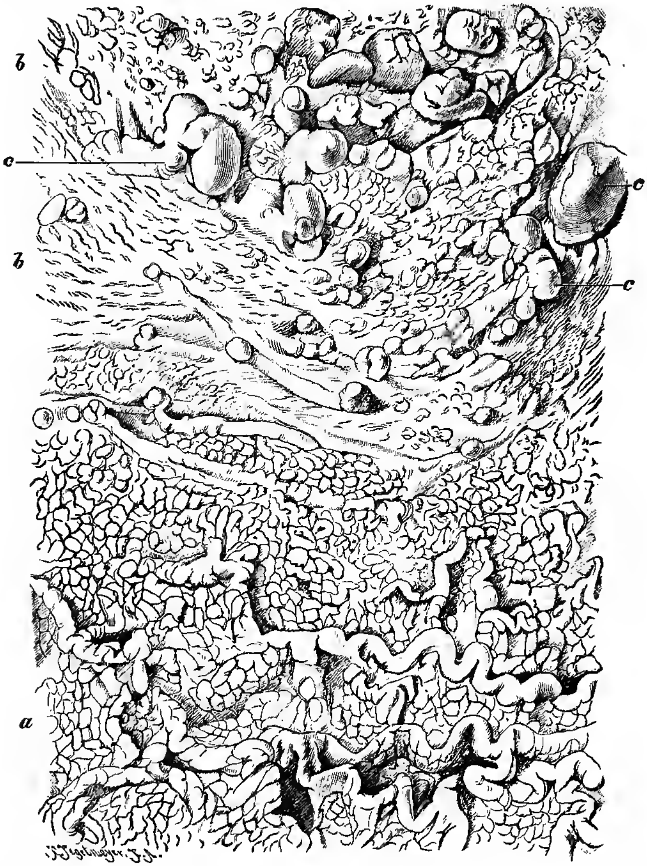

FIG. 322. ATROPHY OF THE GASTRIC MUCOUS MEMBRANE WITH POLYPOSIS (ÉTAT MAMELONNÉ).

(*Natural size*)

a normal mucosa *b* smooth atrophic mucosa *c* polypi

It is not always possible, on post-mortem examination, to determine the nature and origin of such ulcerations, inasmuch as corrosion, chronic catarrh, haemorrhage, and diphtheritic sloughing all give rise in the end to similar appearances.

A not infrequent condition is **atrophy** of the mucous membrane, either simple or associated with other morbid changes, such as cancerous ulceration. Minor degrees of diffuse atrophy are scarcely perceptible by the unaided eye. In severer cases the mucous membrane is sometimes remarkably thin and smooth (Fig. 322 *b*), in others it is beset with small ridges and tuberosities, or larger polypous elevations (*c*), the condition being accordingly described as *polyposis ventriculi* or the *état mamelonné.*

Where the mucous membrane is thin and smooth the glandular structures are more or less effaced (Fig. 323 *a* and Fig. 324 $a\ a_1$), and cases occur in which the membrane over large areas contains no glands at all, or only a few isolated glands (Fig. 324 *a*) some of which are dilated into cysts. In other cases the atrophy of the glandular structures is less marked, the mucosa being reduced to two-thirds or one-half its normal thickness (Fig. 323 *a*). Even when the general atrophy is not extreme, shallow ill-defined ulcerous patches occur here and there in the membrane, within which the glandular layer has entirely disappeared.

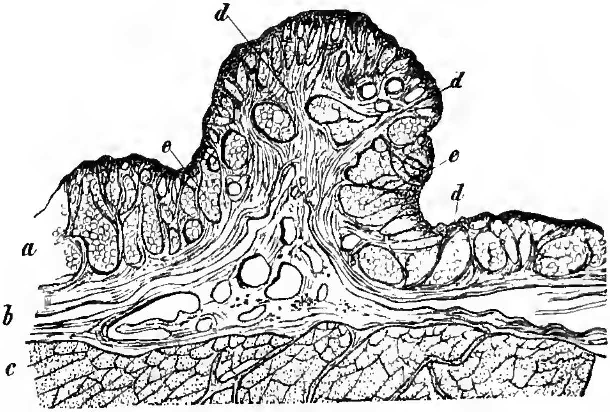

FIG. 323. SECTION THROUGH A SOMEWHAT ATROPHIC AND INDURATED FOLD OF THE MUCOSA (ÉTAT MAMELONNÉ).

(*Preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 8)

a mucosa, with atrophied glands *e* *b* submucosa *c* muscular coat
d hyperplastic fibrous tissue of the mucosa

The connective tissue of the atrophic mucous membrane is more or less altered, and may be hyperplastic both in its deeper and in its more superficial layers (Fig. 323 *d*), so that the condition might be described as one of **atrophic induration.**

Sometimes the mucosa contains foci of small-celled infiltration, and often pigment-granules as well; these give a greyish colour to the inner surface of the stomach. Peculiar hyaline flakes are occasionally deposited in the tissue.

The glandular epithelium is apt to undergo degeneration as the inflammatory process comes to an end. The glands are often seen to be provided, not only at their mouths but in the deeper layers also, with tall cylindrical cells (Fig. 325 *a*), and those that have been dilated into cysts are lined with similar epithelium, many of the cells having the appearance of goblet-cells (Fig. 325 *c*).

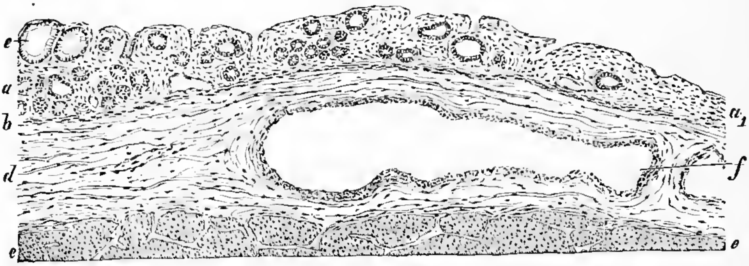

FIG. 324. MARKED ATROPHY OF THE GASTRIC MUCOUS MEMBRANE.

(*Preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam:* × 16)

a a_1 glandular layer of the atrophic mucosa
b *muscularis mucosae*
c dilated glands
d submucosa
e muscular coat
f vein

When the atrophic mucous membrane contains polypous elevations, these consist of more or less altered portions of the mucosa. Within the polypus the connective tissue (Fig. 323 *d*) and the glandular elements are frequently hyperplastic, the latter being moreover atypical in structure and often cystic. The larger cysts are occasionally beset with papillary ingrowths covered with cylindrical epithelium (Fig. 325 *c*).

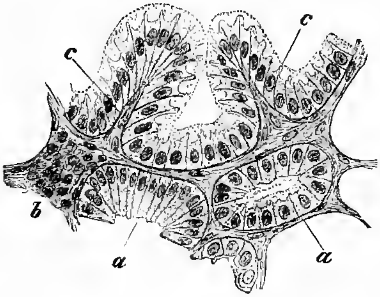

FIG. 325. SECTION THROUGH A GASTRIC POLYPUS.

(*Haematoxylin staining:* × 300)

a tubular gland with cylindrical epithelium
b fibrous stroma infiltrated with leucocytes
c papillary growths into the lumen of a cystic gland

The connective tissue of the submucosa and the muscular coat is in most cases of atrophy but slightly altered; at times however it is somewhat overgrown, and when the inflammation has been chronic it is infiltrated here and there with cells. Marked hypertrophic thickening of the connective tissue of the submucosa and muscular layer is usually a result of cancerous disease (Art. 193). After corrosive ulceration, however, we sometimes find considerable thickening of the layers, usually combined with contraction of the organ as a whole: in very rare cases other inflammatory affections of the stomach also lead to fibroid induration of its walls.

All the forms of fibrous overgrowth are especially apt to affect the pyloric end of the stomach. In the affected area the muscular tissue is usually thickened, but it may become atrophic under the pressure of the cicatricial contraction.

References on Gastritis and Atrophy of the Gastric Mucous Membrane (see also Arts. 188 and 190)

Brissaud: Gastric polyadenoma *A. gén. de méd.* **1885**
Dobrowolski: Lymph-follicles of the stomach *Ziegler's Beiträge* XVI **1894**
Ewald: *Die Krankheiten des Magens* Berlin **1893** (trans. by Saundby) London **1891**
Fenwick: *Atrophy of the stomach etc.* London **1880**
Fischl: Normal and morbid histology of infant's stomach *Prager Z. f. Heilk.* XII **1891**
Hanot and Gombault: Chronic gastritis with hypertrophic sclerosis of the submucosa and indurative retro-peritonitis *A. de physiol.* IX **1882**
Langerhans: Case of chronic proliferous cystic gastritis *V. A.* 116 **1889**
Lewy, B.: Chronic gastritis with atrophy of the mucosa *Ziegler's Beiträge* I **1886**; Case of complete atrophy of the gastric mucous membrane *Berl. klin. Woch.* **1887**
Ménétrier: Gastric polyadenoma *A. de physiol.* I **1888**
Meyer: So-called atrophy of the stomach *Z. f. klin. Med.* XVI **1889**
Nothnagel: Cirrhotic atrophy of the stomach and disappearance of the peptic glands *D. A. f. klin. Med.* XXIV **1879**
Rossoni: Case of cirrhotic inflammation of the stomach with glandular atrophy *Lo Sperimentale* **1883**

190. Inflammations caused by **specific bacteria** and presenting characteristic appearances are upon the whole rare in the case of the stomach, because the peculiar conditions therein subsisting, especially the presence of the acid gastric juice, interfere with the settlement and multiplication of most of the pathogenic bacteria. Manifestations of specific infection are thus exceptional.

The fact that **diphtheria** and **small-pox** on occasion give rise to inflammation of the stomach has already been mentioned (Art. 189).

Infection due to pyogenic micrococci, taking the form of **phlegmonous inflammation,** is rare; both circumscribed and diffuse varieties are however met with, the chief seat of the inflammation being the submucosa.

In the circumscribed variety more or less extensive **abscesses** are formed, which break through into the cavity of the stomach. In the diffuse variety the submucosa is at first greatly swollen and thickened, while the mucosa is unaffected or occasionally somewhat swollen. The exudation in the submucosa is sero-purulent, and gives the tissue a whitish or yellowish-white tint. By the use of proper staining-reagents, the presence of large numbers of micrococci, together with pus-corpuscles (Fig. 326 *a*) and fibrin, can be demonstrated in the affected region. Some of the micrococci at least belong to the genus *Streptococcus* (*c*), and lie partly free in the tissue (*c*) and partly enclosed within the cells (*b*). The mucosa is somewhat infiltrated with round-cells. Sometimes the infiltration extends to the muscular coat, passing chiefly along the intermuscular fibrous septa. In this way the serous coat may come to be affected in like manner. Both muscular and serous coats then become swollen, and the serous surface may be covered with purulent or fibrino-purulent deposits.

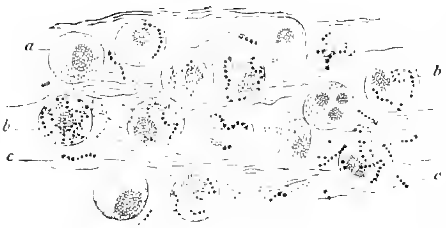

FIG. 326. PHLEGMONOUS INFLAMMATION OF THE STOMACH.

(*Preparation treated with gentian-violet and iodine, and mounted in Canada balsam* : × 500)

a leucocytes
b leucocytes containing streptococci within them
c free streptococci

After a time the submucous tissue appears to break up and dissolve, and the exuded pus breaks at various points through the mucosa. The latter becomes in some cases almost riddled with such perforations. If the patient survive, the smaller openings may cicatrise over and so heal, but the larger frequently remain as cavities extending into the submucosa and ultimately become covered internally with a stratum of epithelial cells: such cavities may communicate with the cavity of the stomach by one or more openings through the mucosa.

Anthrax pustules (Art. 209) and **typhoid** infiltrations and ulcers (Art. 206) are rarely found in the gastric mucous membrane. **Syphilis** and **tuberculosis** also are not commonly manifested in the stomach. In cases of pulmonary tuberculosis, however, gastritis is not infrequent (MARFAN), and is probably in some cases caused by the swallowing of sputum containing the specific bacteria.

References on Specific Bacteria and Gastritis (see also Art. 188).

BARBACCI: A rare variety of gastric tuberculosis *Lo Sperimentale* LXV **1890**
DE BARY: Bacteria in the contents of the stomach *A. f. exp. Path.* XX **1885**
BITTNER: Gummatous gastritis *Prag. med. Woch.* **1893**
CAHN: Diphtheritic gastritis *D. A. f. klin. Med.* XXXIV **1883**
CHIARI: Syphilis of the stomach *Virchow's Festschrift* (*internationale*) II Berlin **1891**
COATS: Tuberculosis of the stomach *Glasgow Med. Journ.* XXVI **1886**
FRAENKEL, C.: Acute emphysematous gastritis of mycotic origin *V. A.* 118 **1889**
KUNDRAT: *Gerhardt's Handb. d. Kinderkrankh.* IV
LANG: *Path. u. Therap. d. Syphilis* Wiesbaden **1885**
LEITH: Phlegmonous gastritis *Edinburgh Hosp. Reports* IV **1896**
LITTEN: Tuberculous ulcer of the stomach *V. A.* 67 **1876**
MARFAN: *Troubles et lésions gastriques dans la phthisie pulmonaire* Paris **1887**

von Recklinghausen: Multiple necroses of the gastric mucous membrane *V. A.* 30 **1864**
Sébillon: Phlegmonous gastritis *Thèse* Paris **1885**
Seitz: *Diphtherie und Krup* Berlin **1877**
Serafini: Tuberculosis of the stomach *Ann. clin. dell' Ospedale degl' Incurabili* Naples **1888**
Smirnow: Membranous and diphtheritic gastritis *V. A.* 113 **1888**
Ucke: Erysipelas of the stomach *Cent. f. allg. Path.* v **1894**

191. After death the mucous membrane of the stomach alters very quickly, the digestive action of the gastric juice being promptly exerted upon the dead tissue. This is especially the case when the stomach contains an excess of gastric juice or of acid products of decomposition. When the wall of the stomach is thus softened, macerated, and dissolved, the condition is spoken of as **cadaveric softening** or **gastromalacia**.

The first alteration consists in solution of the blood-corpuscles and diffusion of their colouring-matter through the tissues, which are thereby tinged of a brownish or greenish-black colour, the acids of the stomach changing the haemoglobin into haematin. Self-digestion follows, and first the mucous membrane and then the muscular and serous coats are transformed into a soft friable pulpy mass, greyish-white, grey, greyish-black, or black in colour, according to the amount of blood present in the vessels. When the stomach is lifted out its contents sometimes break through the softened tissue and escape. Occasionally the walls are thus macerated over their entire extent; and when the fundus has been lying in contact with the diaphragm, the process may extend to the latter and so break it down that the contents of the stomach escape into the thorax. The most marked instances of gastric self-digestion occur in young children, whose stomachs contain a large quantity of milk.

When the contents of the stomach are highly acid, while the circulation within its walls is somehow weakened, the walls may become digested or macerated even during life; though this usually takes place only *in articulo mortis*.

When the circulation is seriously impeded or arrested in any portion of the superficial layer of the mucous membrane, the digestive action of the gastric juice upon it very soon becomes operative, causing solution of the dead or damaged tissue, and producing an **erosion** or **ulceration**. Apart from injuries induced by corrosion and inflammation of the mucous membrane, such grave disturbance of the circulation is most frequently the result of **haemorrhage**, and not only such extravasation as leads to effusion of blood upon the surface, but also that which takes the form of haemorrhagic infiltration of the mucous membrane. Ischaemia from narrowing and occlusion of the arteries occasionally leads to local necrosis of the mucosa, and so to ulceration by erosion.

The gastric mucous membrane is very liable to haemorrhage.

The bleeding may be due to traumatic injury through the swallowing of solid bodies or corrosive poisons, to inflammatory changes in the walls of the blood-vessels, to ulceration, and to venous engorgement such as accompanies portal obstruction in various hepatic and cardiac disorders, scurvy, yellow fever, acute yellow atrophy of the liver, typhoid fever, etc.; in fact to local lesions and to general infective disease, to constitutional disorders and to changes in the composition of the blood as a whole. In rare cases haemorrhage may be the result of primary alterations, such as atheroma and aneurysm, in the large vessels of the stomach and the neighbouring organs.

Copious haemorrhages are observed most frequently in the course of gastric ulcer (Art. 192) and carcinoma (Art. 193), and are due to the erosion and rupture of the larger blood-vessels.

The blood effused on the surface of the stomach, which may be small or large in quantity, becomes rapidly brown or black as the gastric acids transform its haemoglobin into haematin.

The mucous membrane when thus infiltrated with blood is coloured red or brownish-red, or it may be greyish-black. If the haemorrhage is not associated with cessation of the circulation in the infiltrated area, the extravasated blood undergoes the same changes as in any other tissue. Through the formation of pigment the affected spots may assume a greyish colour. Necrosis and digestive solution of the infiltrated tissue leads to a loss of substance which is spoken of as a **haemorrhagic erosion**.

Erosions of the mucous membrane of the stomach are repaired in the same way as those of other mucous membranes, unless some special hindrance stands in the way. Healing takes place by regenerative proliferation, following inflammatory infiltration of the neighbouring tissue. Not only is new connective tissue produced, but the epithelial covering and the glandular structures are restored.

The result is different in certain conditions unfavourable to repair, as when the gastric acids are present in excessive quantity, or when the circulation in the parts around the erosion is interfered with by any cause, local or general (*e.g.* general anaemia). In such cases the circulation in the floor and margins of the erosion may be insufficient to protect the parts from digestion. The exposed strata of the tissue are dissolved one after another, and an ever-enlarging ulcer is produced, the so-called *ulcus ex digestione*. Its enlargement is favoured by the occurrence of thrombosis in the vessels at the base of the erosion.

Melaena neonatorum is a peculiar form of haemorrhage from the stomach and duodenum, which appears in the first week or two of life, and sometimes leads to the formation of ulcers. According to von Preuschen, the haemorrhage is dependent upon cerebral disturbance, that is, on haemorrhages into the brain during birth. Landau seeks to account for it as due to embolic plugging of the arteries of the stomach and intestine. In cases where there is septic infection of the umbilical stump, the haemorrhage may be of septicaemic origin; but such cases are by many (VON PREUSCHEN) not reckoned as examples of genuine melaena neonatorum.

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NEUMANN: Melaena neonatorum *A. f. Kinderheilk.* XII **1891**
VON PREUSCHEN: *Verletzungen d. Kindes bei der Geburt als Ursache der Melaena* Vienna **1894**
REHN: *Centralzeitung f. Kinderheilk.* I
SPIEGELBERG: *Jahrb. f. Kinderheilk.* new series II **1869**

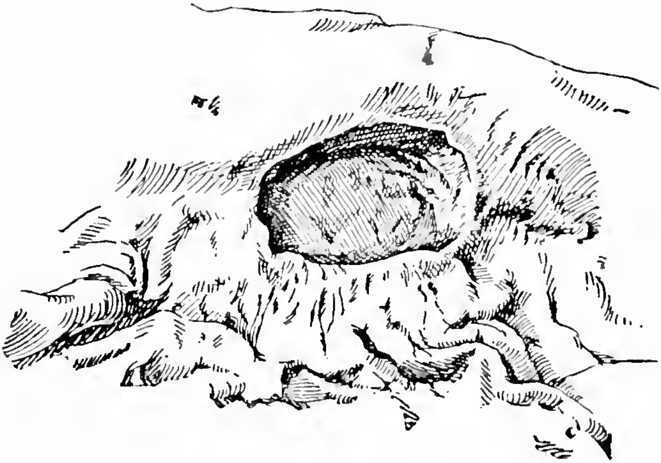

FIG. 327. GASTRIC ULCER.
(*Natural size*)

192. Round or perforating gastric ulcer (*ulcus rotundum, ulcus perforans, ulcus simplex,* or *ulcus ex digestione*). 'Round' ulcer occurs in the stomach and duodenum, and very rarely in the lower part of the oesophagus. As we have said in Art. 191, it is due to the digestive action of the gastric juice, and takes the form of a circumscribed progressive necrosis with solution of the necrosed tissue.

A typical perforating ulcer (Fig. 327) measures from one to six centimetres across, and in form resembles a flat funnel or saucer, inasmuch as the loss of substance in the mucosa or surface layer is greater than that in the upper layers of the muscular coat, and in these greater than in the deeper layers. Where the mucosa rests on the muscular coat a slight ledge or terrace can generally be made out.

In ulcers that are not recent these relations often disappear, the loss of substance in the deeper layers (muscular and serous) becoming as great as in the more superficial ones. Such ulcers may be as much as eight to twelve centimetres in diameter.

When the ulcer extends through all the coats, as it not infrequently does, the adjacent organs, like the pancreas and liver, are usually found to be bound to the stomach by firm adhesions, and so come at length to constitute the floor of the ulcer. In such cases the cavity may become flask-shaped, the hole in the stomach-wall leading into a larger excavation in or bounded by the underlying viscera.

The margins of the ulcer are usually smooth, and not thickened; at most they are slightly swollen. In large ulcers the muscular coat is drawn and puckered beneath the mucous membrane. Outgrowths of atypical epithelium often spring from the mucosa (HAUSER). There is usually no great cellular infiltration of the tissue before it begins to disintegrate. The inflammatory processes which give rise to the adhesions between the stomach and the adjacent organs, and to the thickening of the serous coat, are subsequent to the progressive destruction of the gastric tissue.

Any kind of injury which causes a local textural lesion of the mucous surface of the stomach, and so exposes it to the unchecked action of the gastric juice, may be the originating cause of a gastric ulcer. Probably the commonest of such causes are venous engorgement, haemorrhage, and arterial anaemia due to thrombosis, embolism, spasmodic arterial contraction, arteriosclerosis, or hyaline degeneration of the vessel-walls. In other cases mechanical injury, or corrosion by caustic substances, may afford a starting-point. Perhaps also injurious agencies of a specific nature play some part in the production of these ulcers.

Gastric ulcer runs a chronic course, but the first stages of its development are often somewhat rapid. After severe burns of the skin, for example, ulcers of the stomach and duodenum are sometimes very rapidly induced, probably in consequence of some vascular obstruction or thrombosis due to disintegrated blood-corpuscles.

Round ulcers are most frequently met with in the neighbourhood of the lesser curvature, then near the pylorus, and least frequently in the duodenum.

Even a fully-developed gastric ulcer is capable of **repair.** Complete healing may take place not only in an ulcer limited to the mucosa and submucosa, but even in one wherein all the coats have been perforated. Repair takes place by regenerative proliferation from the adjacent connective and glandular tissues. When the stomach-wall has been entirely perforated, the underlying connective tissue, for example that of the omentum, may unite with new connective tissue growing from the muscular and serous coats, and so fill in the hiatus. By the regenerative growth of the mucosa, the ulcer may not only be closed internally by connective tissue and a covering of epithelium, but a new glandular layer similar to that of the uninjured mucosa may be interposed, though the newly-formed glands are usually atypical in structure.

The muscular fibres appear not to be reproduced, and thus both in the *muscularis mucosae* and in the muscular coat the muscular fasciculi at the site of the ulcer are interrupted by fibrous strands. The cicatrix can accordingly be detected even after the defect is completely made good by the replacement of the glandular and connective structures, and when by contraction of its margins all traces of excavation have disappeared from the internal surface.

Small ulcers may heal without leaving any visible cicatrix. Ulcers of any size give rise to dense puckered scars, which by their contraction lead to considerable deformity of the stomach. A cicatrix near the pylorus is apt to give rise to stenosis of the orifice.

One great danger in gastric ulcer arises from the risk of **haemorrhage** from small or large arteries, which become eroded in the course of the ulcerative process. The haemorrhage is apt to recur again and again and lead to extreme anaemia, or a single copius haemorrhage may bring about death directly. On post-mortem examination it is not rare to find in the floor of the ulcer the eroded vessels, either patent or closed by thrombi, from which the bleeding has taken place. Occasionally one of the larger vessels, such as a main branch of the coronary or splenic artery, is found to have given way.

A still greater danger is that of **perforation** into the peritoneal cavity. This may happen whether adhesions have been set up between the stomach and neighbouring organs or not. In the former case the adhesions are torn asunder, and the contents of the stomach escaping into the abdomen give rise to intense peritonitis.

The organs which adhere to the floor of the ulcer, such as the pancreas or liver, usually exhibit fibrous thickening and hyperplasia at the surface of adhesion. Notwithstanding this, the contents of the stomach may break through into the substance of these organs, and give rise to abscesses in their parenchyma. Sometimes adhesions occur between the stomach and the duodenum or transverse colon. When perforation takes place in such a case a fistulous communication may be opened between the intestine and the stomach. In like manner rupture into the pleural or pericardial sac may occur, and in the latter case leads sometimes to erosion and perforation of the heart-wall itself.

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193. As we have already seen, the connective tissue and the epithelial cells of the gastric mucous membrane frequently undergo proliferous changes leading to the formation of new glandular tissue of atypical structure. This occurs both in the course of atrophic affections of the mucous membrane (Art. 189) and at the margins of healing ulcers, and not infrequently results in the production of polypous excrescences which are spoken of as **glandular polypi.**

Atypical epithelial proliferations which lead to the formation of infiltrating epithelial growths, in other words to **carcinoma,** are very common. They may start in apparently normal mucous membrane, or in membrane that is already morbidly altered and atrophic or ulcerated. The neoplasm starts in the mucosa (Fig. 328 *a*), but very quickly extends to the submucosa (*b*): in this way it is frequently found that the main mass of the growth lies beneath the mucosa in the submucous stratum. Thence the disease invades the muscular (*c*) and the serous (*d*) coats.

In the latter it usually spreads in the form of discrete nodules and nodes, which are distinctly perceptible from without, and follow the course of the lymph-vessels. At a later stage it may invade the veins, giving rise to carcinomatous thromboses, which take the form of long flat elevations of the serous surface, lying chiefly about the pyloric end of the stomach.

The lymph-glands situated behind the lesser curvature soon become notably enlarged, and are often transformed into huge cancerous nodes. The disease may likewise extend to the omentum and give rise to a general thickening of its tissue or to irregular tuberous growths. In other cases metastases occur in the peritoneum generally, and in the liver, lungs, etc. The liver is the commonest seat of the metastatic growths, owing to the distribution in the stomach of the tributary branches of the portal vein.

Gastric carcinoma most frequently takes the form of soft elevated fungous tumours arising from the pyloric end and along the lesser curvature (Fig. 329): tumours arising from the fundus or cardiac end, and general or diffuse cancerous infiltration, are more rare. When the tumour projecting into the cavity of the stomach has attained a certain size, its central parts usually break down, and a carcinomatous ulcer is produced (Fig. 330 *b*). Such an ulcer is distinguished by its raised rampart-like borders. Its floor is generally formed by the submucosa, which is thickened by fibrous hyperplasia or cancerous infiltration. The fibrous tissue of the muscular and serous coats is often the seat of extensive hyperplasia, or is infiltrated with cancer-cells, so that the whole pyloric region appears thickened and indurated. The stage at which destructive processes start in the growth is very different in different cases. It may thus happen that in one case the growths in the interior of the stomach (Fig. 329) are large and

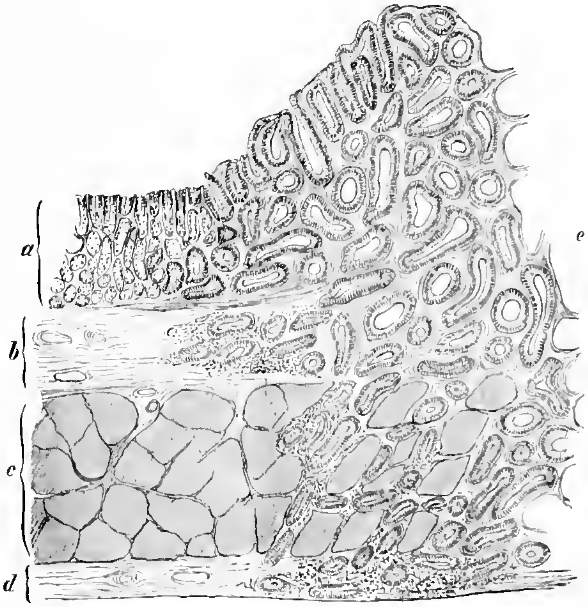

FIG. 328. ADENO-CARCINOMA OF THE STOMACH.

(Diagrammatic: preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam: × 25)

a mucosa *b* submucosa *c* muscular coat *d* serous coat *e* neoplasm wide-spreading, forming bulky fungous and polypous tumours, while in another case even minute growths have already begun to ulcerate. Cases indeed occur in which no definite projections are produced at all, and the process from the outset consists essentially in cancerous infiltration of the submucous and other coats, which quickly extends over a considerable area.

Occasionally the neoplastic tissue within the cavity of the stomach becomes entirely disintegrated, so that the surface of the resulting ulcer appears smooth and level. When in such a case the submucous, the muscular, and the serous coats are not visibly beset with nests and clusters of cancer-cells, but are simply indurated in consequence of fibrous hyperplasia, the affected region assumes the look of a simple non-malignant or fibroid induration. Cases occur in which no cell-nests are to be made out even when the tissue is microscopically examined, and then the only evidence forthcoming as to the cancerous nature of the disease may be the metastatic growths to which it has given rise.

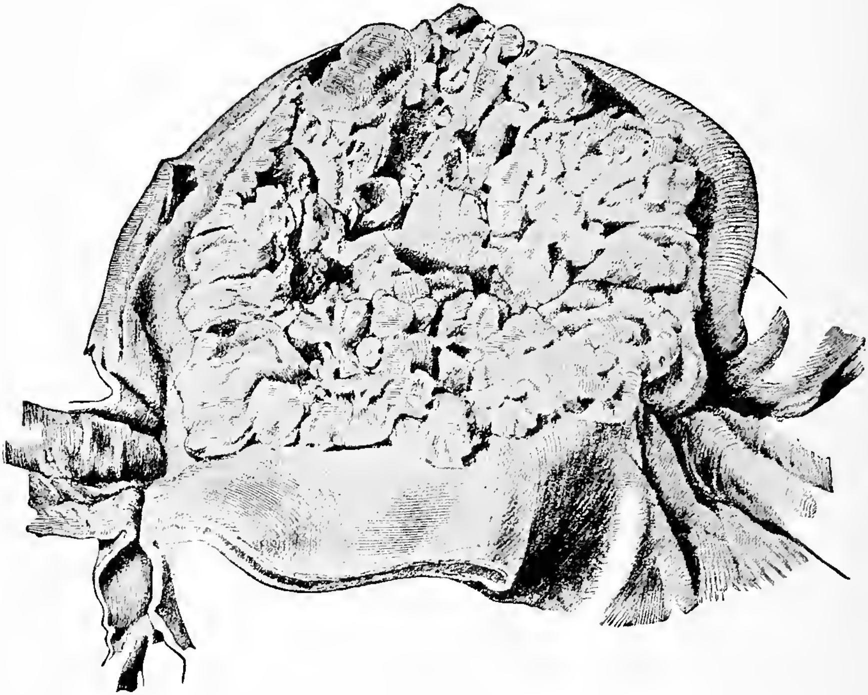

FIG. 329. FUNGOUS CARCINOMA OF THE STOMACH IN THE NEIGHBOURHOOD OF THE PYLORUS.

(*Five-sixths of the natural size*)

Five chief forms of gastric cancer are distinguished according to their histological structure.

(1) **Medullary carcinoma** (Fig. 329) takes the form of soft fungous excrescences or low rounded swellings, chiefly about the pyloric end of the stomach. As the central parts break down these growths give place to ulcers with raised borders, that are white and pulpy in appearance. The new-growth starts in the gastric glands. Structurally it is distinguished by the presence of an extraordinary number of cell-nests or loculi, while the stroma is but slightly developed. After the destruction of the new-growth fibrous induration of the muscular wall very frequently takes place. This form produces many metastases.

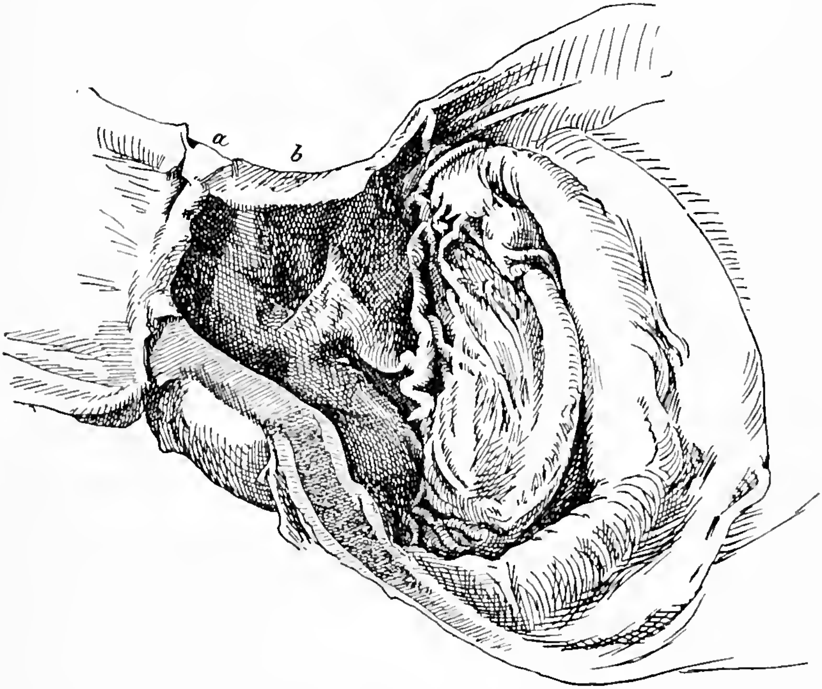

FIG. 330. CANCEROUS ULCER WITH INDURATION OF THE PYLORIC END OF THE STOMACH.

(*Three-fourths of the natural size*)

a pylorus *b* cancerous ulceration

(2) **Destructive adenoma** (*adeno-carcinoma, carcinoma medullare adenomatosum,* or *epithelioma*) also gives rise to soft nodular growths, which presently break down and ulcerate. The neoplasm is distinguished by the presence of atypical tubular glandlike structures, often possessing a simple cylindrical epithelium

(Fig. 328 *e*), from which by the continued production of new cells solid cellular columns are finally built up. The stroma is not abundant, and is often infiltrated with leucocytes.

(3) **Scirrhous cancer** or fibroid carcinoma appears in the form of diffuse thickening and induration of the stomach-wall, especially the pyloric part of it (Fig. 330). The pylorus itself is usually more or less constricted. The inner surface of the diseased region is covered partly with mucous membrane, partly with the exposed and indurated fibrous tissue belonging to the submucosa. On section the various coats are distinguishable, but each is more or less thickened by fibrous hyperplasia. It is probable that so-called scirrhus is often nothing but an induration of the stomach-wall, partly cancerous and partly fibroid, left as a secondary result of the ulcerative disintegration of a soft cancer.

(4) **Colloid or gelatinous cancer** takes the form both of nodular swellings and of diffuse and wide-spread infiltration of the stomach-wall. In each form the neoplasm contains patches of transparent jelly-like appearance, or consists almost entirely of colloid substance. The growth may spread to the peritoneum, and there speedily gives rise to large semi-transparent colloid growths, which are more or less richly supplied with blood-vessels. On microscopic examination it appears that the colloid masses are partly derived from the cancer-cells, partly from the fibrous stroma of the growth.

(5) **Squamous epithelial cancer** is rarely met with in the stomach. It affects the cardiac end and the neighbouring parts of the oesophagus.

The **connective-tissue growths** of the stomach have very little pathological interest. A few cases of nodular sarcoma, and of lipoma, fibroma, and myoma have been recorded. The tumours usually project into the cavity of the stomach, seldom appearing externally.

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CHAPTER LXIV

THE INTESTINE

194. **Congenital deformities and misplacements.** Absence of the whole or of a large part of the intestine is met with only in very ill-developed acardiac monsters. Minor deficiencies, constrictions (Fig. 331 *b c d e*), occlusions, and diverticula are somewhat commoner.

The anal region is that which is oftenest imperfectly developed. The allantoid cloaca may persist, that is to say the intestine and bladder open into a common chamber or orifice. In such cases the bladder is frequently unclosed and the lower bowel absent, so that the ileum communicates directly with the cloaca. In less-marked cases there is merely an imperfect separation of the rectum from the urogenital sinus, into which latter the genital and urinary canals open in the foetus. The anus itself, which is developed from an invagination of the external skin, is wanting, and the condition is described as *atresia ani* or **imperforate anus**: and according as the lower end of the bowel is connected with the bladder, the urethra, or the vagina, the atresia is distinguished as vesical, urethral, or vaginal. When the rectum is completely separate from the urogenital sinus, although not in communication with the anal invagination, we have simple *atresia ani*.

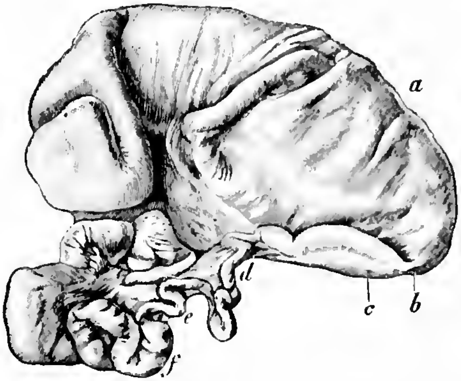

FIG. 331. HYPOPLASIA OF THE SMALL INTESTINE OF AN INFANT.

(*Five-sixths of the natural size*)

a	widely-dilated portion
b c d e	contracted and ill-developed portion
f	normally-developed portion

The formation of abnormal septa in the continuity of the intestine is rare.

A frequent malformation is the so-called **Meckel's diverticulum.** This is a cylindrical or flask-shaped appendage attached to the ileum about one metre or more above the ileo-caecal valve. It is a remnant of the omphalo-mesenteric duct. In rare cases it is connected by a cord with the umbilicus, and in still rarer cases it opens on the exterior, just below the umbilicus. Cases occur in which abstricted portions of the diverticulum form tumour-like masses in the navel, constituting the so-called umbilical adenoma. These growths correspond in their structure to that of the diverticulum, and are composed of the same elements as the intestinal wall.

Other diverticula and dilatations of the intestine are met with, particularly in the large intestine; when faeces accumulate in them they occasionally attain a very considerable size.

A piece of intestine lodged in a sacculation of the peritoneum is spoken of as a **hernia** (Art. 195), and a coil or other portion which escapes to the exterior through an opening is called a **prolapse.**

Owing to the fixed position of certain points in its course, **displacements of the colon** are the most easily recognised of the malpositions of the intestine within the abdominal cavity. The caecum, for instance, varies much in position; it may lie either below or above the line joining the anterior superior spines of the ilia. The level of the hepatic and splenic flexures differs much in different persons. The length of the sigmoid flexure and of the transverse colon is very variable. In some cases the latter may be almost absent, the ascending and descending portions lying side by side on the right of the abdomen.

Enterocystomata (ROTH) may be combined with other malformations of the intestine. They are closed sacs filled with liquid, and exhibiting the same structure as the walls of the bowel. Two forms may be distinguished—(1) cysts due to closure of an otherwise normally-developed intestine; (2) cysts due to some abnormality of development in the foetus. The supernumerary parts of the intestine, usually dilated into one or more cysts, may in reality be portions of the intestine of a rudimentary twin, and therefore teratoid in character, or depend on the closure and abstriction of an anomalous diverticulum, such as Meckel's. They are apt to increase greatly in size from accumulated secretion, and then become displaced from their original site, lying either in the abdominal cavity or lodged in the wall about the umbilicus.

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195. **Acquired deformities and displacements. Abdominal hernia** or 'rupture' in the stricter sense implies the entrance of some part of the normal abdominal contents into a sacculation of the peritoneum which either protrudes to the exterior or remains lodged within one of the cavities of the body.

In **external hernia** (Fig. 332 *a c*) some viscus which is covered with peritoneum escapes from the abdomen, pushing before it the subperitoneal structures and the skin, the escape taking place either through some normal opening that has become abnormally dilated or through some abnormal opening.

The protruding viscus accordingly lies in a sacculation of the parietal peritoneum (*a c*), which forms the **hernial sac.** It can be absent only when the peritoneum has been torn by traumatic injury, or when the displaced viscus is extraperitoneal (such as part of the bladder or caecum) and protrudes directly through some opening in the fascia or muscles of the abdominal wall (prolapse).

The other tissues that are forced outwards with the hernia are spoken of as the **accessory coverings** of the hernial sac. The inner layer or covering consists of the subperitoneal (subserous) cellular tissue, which is usually thickened and toughened (peritoneal fascia). In femoral and inguinal hernia the true fascia (*fascia propria, b d*) comes next, and is continuous with the fibrous structures bounding the orifice in the abdominal wall through which the hernia has escaped.

At first the hernial sac is simply subglobular or saucer-shaped; when fully developed it is in general flask-shaped. The place where the viscus passes externally through the fascia and muscles is spoken of as the hernial orifice or **ring.** The narrower part of the sac where it is gripped by the structures of the orifice is called its **neck.** The peritoneum is drawn into radial folds as it passes towards the neck of the sac.

The **contents of a hernia** may be any of the various viscera of the abdomen. Most commonly they consist of a part of the omentum or small intestine, less commonly the caecum or colon, and still less commonly other viscera, such as the ovaries, bladder, stomach, liver, etc. Very large ruptures, such as occur in the inguinal region, may include the greater part of the bowels. When a portion only of the intestinal wall or Meckel's diverticulum is included in the hernial sac we have a case of what is called **Littre's hernia** (A. LITTRE: *Mémoires de l'acad. royale* Paris **1700**).

In many hernias the sac is already formed before the viscera escape. Thus inguinal hernia is produced in children by the passage of the intestine into the persistent vaginal process of the peritoneum (infantile or congenital hernia). Hernia may also arise in consequence of some external stretching force by which the peritoneum is dragged outwards. Thus a lipoma growing in the septum crurale, and pressing outwards as it increases in

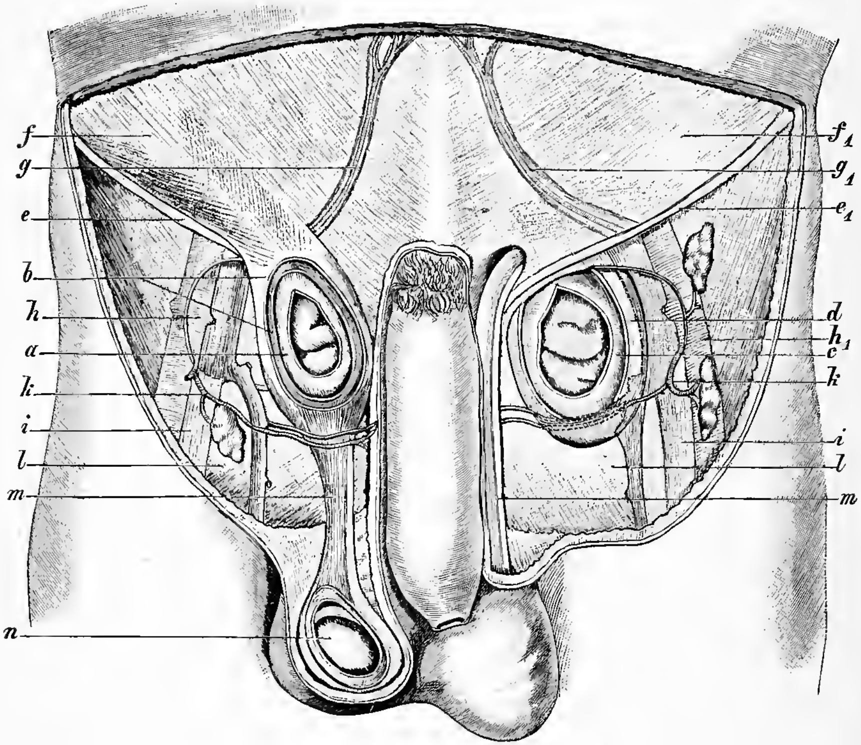

FIG. 332. RIGHT INGUINAL HERNIA AND LEFT FEMORAL HERNIA.
(*After* JOESSEL)

a hernial sac of the inguinal hernia with loop of intestine
b fascia propria
c hernial sac of the femoral hernia
d fascia propria
e e_1 Poupart's ligament
f f_1 aponeurosis of the external oblique muscle
g g_1 epigastric vessels
h femoral artery
i femoral vein
k external pudic artery
l fascia lata (superficial layer)
m spermatic duct
n testis

size, sometimes drags upon and sacculates the peritoneum which is firmly attached to the septum. Lastly, some local diminution of the resistance of the abdominal wall may in certain situations cause the peritoneum to become sacculated under the action of the expiratory pressure.

Inguinal hernia (Fig. 332 *a*) takes place in the groin, and is due either to the congenital patency of the vaginal process of the peritoneum after descent of the testis, or to a secondary protrusion of the peritoneum in the inguinal canal.

In **oblique** or external inguinal hernia, the neck of the sac passes down the canal from the internal to the external ring: in **direct** or internal inguinal hernia the peritoneum is pushed from within directly through the external ring. The orifice of an oblique hernia lies externally to the epigastric artery, while that of the direct variety is to the internal side of the artery. Inguinal hernia may reach a large size and contain the greater part of the bowels. It is the commonest of all varieties, especially in men.

Femoral or crural hernia (Fig. 332 *c*) is due to the protrusion of the peritoneum beneath Poupart's ligament (e_1) through the opening traversed by the great femoral vessels. It is a common variety, especially in women.

In **obturator hernia** (Fig. 333) the sac passes with the obturator nerve and artery through the obturator or thyroid foramen of the innominate bone. This variety is rare.

In **ischiatic hernia** the protrusion is through the ischiatic notch beneath the glutaeus maximus: it is also rare.

In **perineal hernia** the sac escapes between the anterior fibres of the levator ani: it is an uncommon variety.

Umbilical hernia is either congenital, consisting of a protrusion of the intestine into the stump of the cord, or acquired, and is then due to the escape of a piece of the intestine or omentum through the dilated umbilical ring. The acquired form is most commonly met with in women who have borne many children.

Ventral hernia is due to the general relaxation or stretching of the fibrous structures of the front of the abdominal wall, the peritoneum protruding between the muscles which are thereby thrust asunder.

Internal hernias are produced by the incarceration of coils of intestine or other abdominal viscera in peritoneal pouches, that either lie within the abdominal cavity or protrude into the thorax.

Among such pouches we may mention the lesser or omental sac of the peritoneum bounded by the stomach, pancreas, liver, and spleen, and communicating with the greater sac through the foramen of Winslow, between the hepato-duodenal and duodeno-renal ligaments; the duodeno-jejunal fossa, between the upper part of the mesentery of the small intestine and the spine; the subcaecal fossa, on the mesial side of the caecum; and the intersigmoid fossa, beneath the mesocolon of the sigmoid flexure. Coils of intestine may slip into and be incarcerated in any of these pouches. The duodeno-jejunal fossa may indeed enclose the whole of the small intestine (retroperitoneal hernia).

Hernias protruding into the thoracic cavity are spoken of as **diaphragmatic hernias**: they occasionally reach a considerable size.

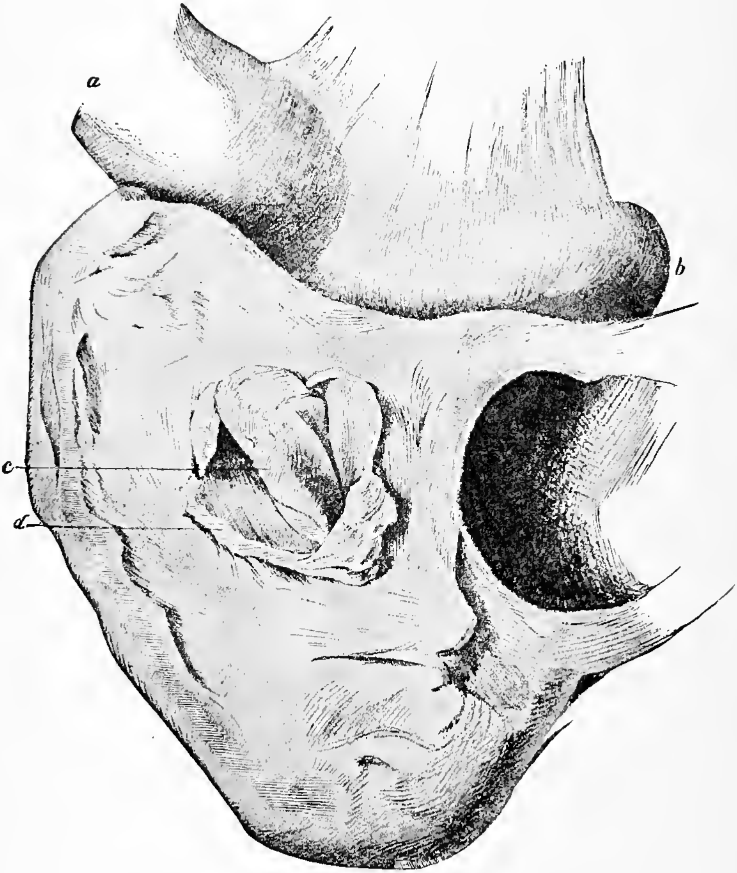

FIG. 333. INCARCERATED OBTURATOR HERNIA.

a intestine entering the sac *b* intestine leaving the sac *c* intestine within the sac *d* hernial sac

196. When a hernia is once established, further changes in the parts usually follow. The hernia may increase in size by the inclusion of more of its abnormal contents. The sac stretches and becomes thinner, or new portions of the peritoneum are dragged into it.

In consequence of the constant mechanical lesions to which the hernia is exposed, or from specific infection (as in tuberculosis), a certain amount of inflammation is usually set up about it. The sac thereby thickens, and the folds of the peritoneum at the hernial orifice cohere so that the channel of communication between the sac and the abdominal cavity becomes thickened and inextensible. The serous covering of the included intestine and its mesentery, and the omentum, become thickened in like manner. Lastly, adhesions may be set up between the different parts of the sac, or between different loops of intestine and the sac-wall. The omentum is especially apt to adhere to the sac.

These changes very rarely indeed lead to cure by the closure and obliteration of the empty sac; on the contrary they usually make matters worse. The thickening and adhesions of the serous surfaces diminish by degrees the mobility of the included viscera. The neck of the sac becomes narrower, constricting its contents more and more. At length the contents can no longer be returned to the abdomen, and the originally reducible hernia has become irreducible.

When the contents of a hernia (reducible or not) are so constricted or compressed that the included intestine becomes impervious and its circulation seriously impaired, the hernia is said to be strangulated or incarcerated. **Strangulation** may also be due to the overfilling of the intestinal coils with faeces (faecal incarceration), whereby the coils entering into the sac become distended and compress or acutely flex the efferent coils. When the sac is thus distended by the overfilling of the intestine the compression of the coils at the neck is naturally increased.

A further form of incarceration occurs when the orifice, by the mere elasticity of the surrounding tissue, strangulates a loop of intestine which has escaped through it (elastic incarceration). This happens, for example, when the intestine is suddenly forced through the opening by some increase of pressure within the abdominal cavity and the ring is thus forcibly dilated beyond its normal size. Lastly, the intestine may be strangulated by bands of adhesion within the sac; or a second coil of intestine may enter where there has previously been but one, or a piece of omentum may be squeezed into the neck of the sac and thus compress the intestine, and so on.

When a coil of intestine or a part of the omentum is constricted and strangulated, disturbances of its circulation take place. The venous efflux is impeded, and engorgement, transudation, and haemorrhage result. The coil becomes purple and swollen, while liquid gathers in the sac; and these factors conspire to intensify the strangulation.

If the incarceration is not relieved the intestine sooner or later becomes gangrenous, and intense inflammation of the hernial sac ensues. The intestine becomes discoloured, turning brown or bluish-black; at the point of strangulation it is usually paler and somewhat greyish. Presently perforation takes place, and at the boundary between the living tissue and the dead, that is at the inner border of the strangulating constriction, suppurative inflammation is set up.

197. When inflammatory false membranes and adhesions exist within the abdominal cavity, so as to form abnormal foramina or pouches, the intestine may be caught in these, and thus undergo **internal incarceration** (Fig. 334). If the obstruction is severe and persistent, the same changes take place in the affected coils as occur in strangulated hernia. The like may happen when the omentum or the mesentery possesses abnormal perforations or clefts through which the intestine can slip.

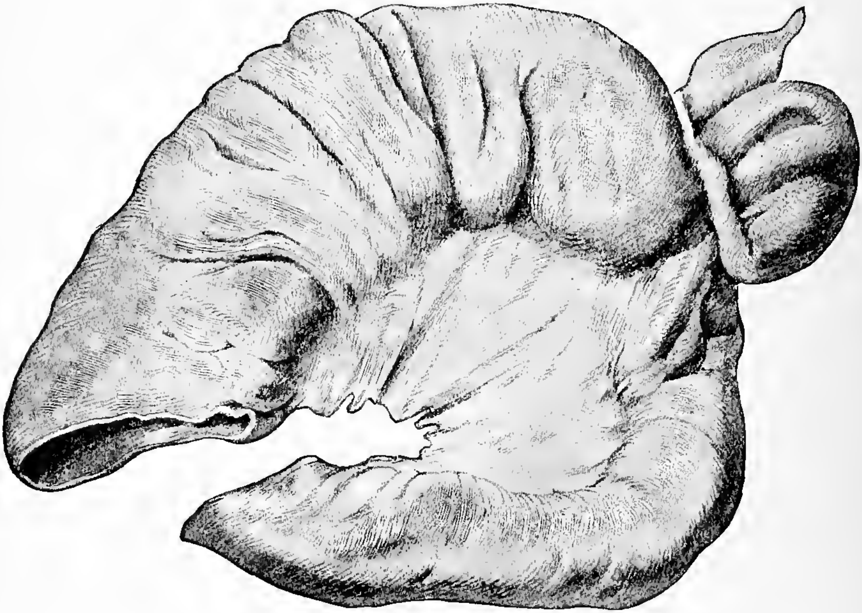

FIG. 334. INTERNAL INCARCERATION.

(*A portion of the small intestine has slipped between adhesive bands connecting a caseous lymph-gland and the intestinal wall*)

Another cause of intestinal obstruction is twisting of the bowel on itself, or **volvulus**. It can occur only in the movable parts of the tract, and is due partly to peristaltic movements, especially when the tube is very unequally distended, and partly to external violence, such as a blow on the abdomen. The twist occurs at the mesenteric attachment, the two limbs of the coil crossing each other over the mesentery. The channel of the intestine is occluded and the mesenteric circulation stopped.

Untwisting is prevented by the weight of the distended coil and the pressure of the rest of the abdominal contents.

Twisting of the sigmoid flexure or of the small intestine may result in a kind of knotting between the two, the attached end of the twisted coil of one part becoming encircled by a loop of the other.

A not uncommon kind of displacement or dislocation of the intestine is that known as **intussusception** or invagination (Fig. 335). In this a higher part of the bowel slips into or is invaginated by a lower; rarely the reverse. Recent intussusceptions are most frequently met with in the small intestine in young children, especially in those who have died of cerebral or intestinal affections.

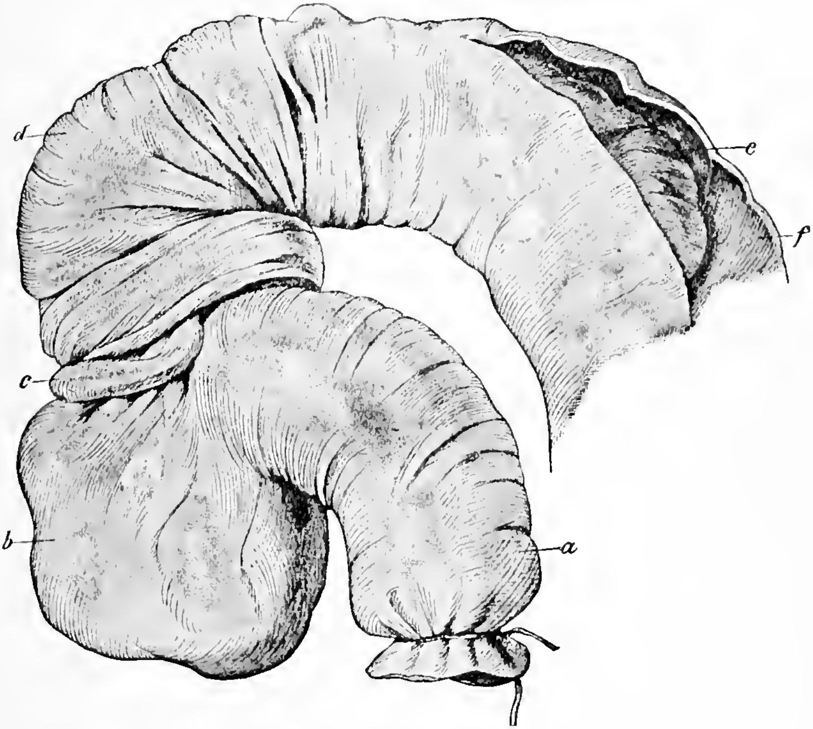

FIG. 335. INTUSSUSCEPTION OF THE INTESTINE AT THE ILEO-CAECAL VALVE.

a ileum
b caecum
c vermiform appendix
d transverse colon
e invaginated and everted coil of intestine
f descending colon

The extent of the invagination varies much. Where the intestine is very loose and movable a very considerable length of it may be involved. Such extreme cases of invagination occur especially in the region of the ileo-caecal valve (Fig. 335), whereby not only the small intestine but also the upper portion of the large intestine, with the caecum, may enter the lower portions of the colon, until finally the invaginated part of the bowel reaches the sigmoid flexure or even the rectum.

When the intussusception remains unreduced, the mesentery being severely dragged upon and its vessels compressed, the invaginated portion of the bowel becomes highly hyperaemic and oedematous, and ultimately undergoes necrosis and gangrene. Presently inflammation is set up, and this may lead to adhesions between the enclosed and enclosing segments. When the necrosed portion of the invaginated intestine is separated so as to be cast off entirely, recovery sometimes takes place by the adhesion of the upper and lower segments. This however is a rare result, and the patient usually perishes from the gradual occlusion of the canal and the consecutive inflammation.

The cause of intussusception is not certainly made out. According to LEICHTENSTERN it depends on paresis or partial paralysis of a limited portion of the bowel. When this occurs an energetic peristaltic movement of the portion immediately above may thrust the latter into the flaccid or paralysed portion.

Prolapse of the bowel implies its direct escape through some normal or abnormal opening. The anus is the only possible normal opening to be considered, and through it the rectum may protrude. *Prolapsus recti* (or *ani*) occurs during violent straining at stool, especially when the intestine is relaxed by chronic inflammation. The protruding bowel forms a kind of tumour covered with mucous membrane; it often becomes inflamed or gangrenous, particularly in cases where by adhesions or constriction the prolapse has become irreducible. Prolapse through abnormal openings occurs mainly in the case of perforating wounds of the abdomen and of traumatic rupture of the diaphragm.

Stenosis and **atresia** are not infrequently the result of inflammation of the wall of the intestine itself. The serous coat may become inflamed, and so indurated, cicatrised, and contracted that the lumen of the canal is obstructed; or ulcerative inflammation of the mucous membrane may result in the formation of new fibrous tissue, which subsequently contracts. Carcinomatous tumours very frequently lead to stenosis, either by protruding into the lumen of the intestine or by ulceration resulting in induration and contraction of its wall (Fig. 352 *a* and Fig. 353).

Dilatation of the intestine is most commonly due to retention of faeces or flatus, from stenosis or invagination of the lower portions or from abnormal relaxation of the intestinal walls.

Usually all the coats are dilated together; sometimes however the muscular structures are here and there pushed aside, and the mucous and submucous coats bulge through laterally, producing sacculations or **false diverticula** of various sizes.

Sacculations are also sometimes due to the local yielding of the wall in its whole thickness.

Perforation of the bowel is in general due to some local textural change, especially to ulceration of the mucous surface, necrosis, or softening occasioned by suppuration in the neighbourhood of the part. Perforation may of course be caused by mechanical violence.

The result of perforation, unless the lips of the wound immediately close, is local or general peritonitis from the escape of faeces into the abdominal cavity. The peritonitis is local when by previous inflammation adhesions have been set up around the point of perforation. If faeces escape into the portion of the peritoneum so circumscribed a **faecal abscess** is produced; this may break either outwardly or into another part of the intestine.

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198. **Disturbances of the circulation** in the intestine, resulting in hyperaemia or anaemia, are of common occurrence, though the post-mortem appearances usually give but an inadequate idea of the conditions as they existed before death.

Congestive hyperaemia, indicated by intense redness of the mucous membrane, is at times so extreme as to lead to haemorrhage. It is due either to something of an irritating nature in the intestinal contents or to some disorder of the vaso-motor innervation, reflected it may be from a remote point. General **passive hyperaemia** accompanies general engorgement of the major circulation, diseases of the liver which cause partial interference with the portal circulation, and obstruction or occlusion of the portal vein itself. The engorgement in these cases is however not so well marked in the intestine as in the stomach. Local venous engorgement results most frequently from compression of the mesenteric vessels pertaining to the intestinal coils lying in a hernial sac or otherwise constricted and compressed; it is rarely due to thrombotic occlusion of the mesenteric vessels. Interference with the efflux of blood in combination with continued afflux often gives rise to a purple or dark-red coloration not only of the mucosa but of the serous and muscular layers.

Haemorrhage may be a result both of active and of passive hyperaemia; it is commonest on the mucous surface, the villi readily permitting the blood to exude. Haemorrhage is a frequent accompaniment of various inflammatory affections, such as dysentery and typhoid fever. It occurs both at the onset of inflammation and as a consequence of ulcerous destruction of the mucous membrane. The effusion may be limited to a single spot or to a few scattered areas, or it may be widely diffused. Intestinal bleeding from congenital or acquired haemophilia, or from vaso-motor paralysis, sometimes extends over the larger portion of the intestinal tract. The blood poured out into the intestine assumes after a time a tint varying from brownish-black to black, and usually becomes semi-liquid or 'tarry.' The blood extravasated into the mucous membrane gives rise to slate-coloured or black patches, which persist for a long while. Dense haemorrhagic infiltration of the mucous membrane, with the accompanying arrest of the circulation, ends in necrosis of the tissue. The dead part is by and by exfoliated, and a loss of substance results.

Extreme local disturbances of the circulation, such as arise from compression and thrombosis of the veins, or from embolic and thrombotic occlusion of the superior mesenteric artery and its branches, sometimes induce not only haemorrhagic effusion into the mucous membrane, but haemorrhagic infarctions of the entire thickness of the bowel-wall. The affected coils assume a bluish-black appearance, and then undergo necrosis from arrest of the circulation.

Oedema of the bowel is a consequence of venous engorgement or of damage to the vessel-walls, such as usually accompanies inflammation. Oedema of the submucosa sometimes gives rise to considerable swelling of the intestinal wall, the mucous membrane at the same time having a sodden and dropsical appearance.

Atrophy of the mucous membrane, characterised by the disappearance of its glandular elements, is very common, especially in the large intestine and about the caecum. It is usually a result of antecedent inflammation (Art. 199). **Amyloid degeneration** of the connective tissue of the blood-vessels is the most important of the degenerative changes met with in the intestine. It occurs chiefly in the mucosa and submucosa, more rarely in the muscular layers. It makes its appearance under the same conditions as amyloid degeneration of the glandular organs, but in rare instances its origin cannot be traced. Mucous membrane which has undergone this form of degeneration is usually pale: when extreme it gives rise to induration.

The muscular coat of the intestine is rarely the seat of any striking change, though the muscle-cells are subject to various forms of degeneration, fatty, hyaline (with the formation of homogeneous translucent flakes), and pigmentary. In advanced age the muscle-cells of the ileum very often contain yellowish pigment-granules containing no iron, which sometimes give to the intestine a yellowish-brown tint that is apparent even to the unaided eye. In infective diseases, in certain toxaemic and cachectic conditions, and in disorders of the central nervous system, degeneration of the nerve-plexuses of the intestine has been observed to take place (SCHEIMPFLUG, BLASCHKO, and SASAKI).

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199. Most **inflammations** of the intestinal tract are caused by changes in its contents; though matters reaching it by way of the blood and lymph, and toxic and infective agents such as arsenic, corrosive sublimate, and septic poisons, also give rise to exudations into the canal and inflammatory changes in the walls (Fig. 39).

The intestinal contents are liable to exert an irritative and injurious action simply by remaining too long in the intestine, and so giving opportunity for the bacteria, which are always present there, to induce abnormal decompositions. This is most apt to happen in the large intestine, where a certain amount of putrefactive decomposition normally occurs; but it also takes place in the small intestine, where abnormal, generally acid, fermentations may be set up. These decompositions are as a rule due to the action of the normal bacteria of the intestine, but special kinds are not infrequently ingested with the food. Some of the inflammatory affections of the bowel ascribed to unwholesome meat, sausages, or cheese, are referable to abnormal putrefactive decompositions dependent on the action of certain bacteria swallowed with the damaged articles of food. In other cases bacteria are introduced which not only multiply in the contents of the bowel, but also settle and grow in its tissues, inducing therein grave pathological changes. In others again poisonous chemical products of bacterial decomposition, and poisons elaborated by other plants or animals, are ingested with food or drink. Lastly, there are many chemically and physically active substances, such as corrosive poisons, which are capable, when they reach the bowel, of inducing inflammation in it (Art. 188).

Catarrhal enteritis or intestinal catarrh is the commonest form of inflammation of the bowel. The catarrhal secretion may be serous, mucous, purulent, or of some intermediate kind. In acute catarrh the mucous membrane is hyperaemic and swollen, the tissue more moist than normal, and often more or less infiltrated with leucocytes, while here and there blood may be extravasated. In the severer forms the submucosa contains exuded liquid and leucocytes. The epithelial cells pour out an excessive quantity of mucus, and large numbers become detached. The reddened mucous membrane is accordingly covered with a slimy film or with turbid liquid. According to BÖHM excessive desquamation is characteristic of poisoning by arsenic and by toxic substances of the muscarin-group, though many other varieties of catarrh are accompanied by great shedding of the epithelium; this is especially marked in the case of cholera. In the chronic affection known as **membranous enteritis** the production of mucus is enormous, and the epithelium desquamates freely, tube-like casts and membranous masses being passed by the bowel.

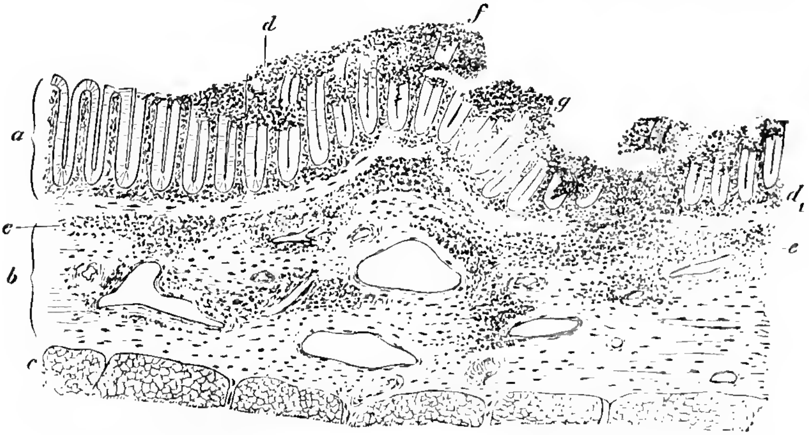

FIG. 336. SUPPURATION AND NECROTIC DISINTEGRATION OF THE MUCOUS MEMBRANE OF THE COLON.

(*From a case of dysentery: haematoxylin staining*; × 25)

a mucosa
b submucosa
c muscularis
d interglandular, and
d_1 subglandular infiltration of the mucosa
e infiltration of the submucosa
f superficial glandular layer, infiltrated and exfoliating
g ulcer, the floor of which is infiltrated

Most cases of catarrh run an acute course, and end in recovery, though permanent structural changes are frequently left behind. Chronic catarrh is generally due to the continued introduction of irritating substances, to abnormal decomposition of the intestinal contents, or to engorgement of the portal vessels.

When the catarrh assumes a purulent or muco-purulent character, so that the surface of the mucous membrane is covered with pus or muco-purulent secretions, the accompanying infiltration of the mucous membrane is usually very dense (Fig. 336 $d\ d_1$), and in places may be so abundant that the glandular structure is entirely concealed by it. Under these conditions partial suppuration of the mucous membrane is apt to supervene, so that at certain spots the tissue perishes and exfoliates (*f*), and more or less extensive **ulcers** (*g*) surrounded by zones of cellular infiltration are formed. The shreds of dead tissue often form a furfuraceous coating, which together with the exuded pus overlies the greatly swollen and reddened mucous membrane. The submucosa (*b*) is usually pervaded by multitudes of round-cells (*e*). In rare cases submucous abscesses are formed, which presently rupture and give rise to ulcerous excavations, or wide-spread phlegmonous sero-purulent infiltration of the submucosa takes place.

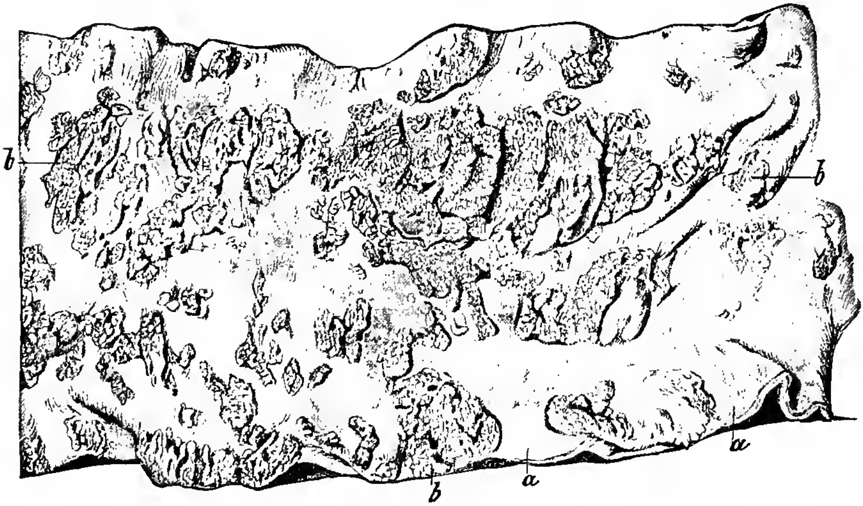

FIG. 337. MUCOUS SURFACE OF THE LARGE INTESTINE AFTER PARTIAL DIPHTHERITIC ULCERATION OF THE MUCOSA (DYSENTERY).

a smooth atrophic and indurated mucosa *b* mucosa with persistent glandular layer

When the necrosis is extensive, the process assumes more and more the character of **diphtheritic inflammation**; indeed, no sharp distinction between the latter and suppuration with molecular disintegration can be drawn. The necrotic tissue lies on the red and swollen mucous membrane (Figs. 343 and 344) in the form of dirty-grey, slate-coloured, or yellowish sloughs; these are mainly found on the ridges of the intestinal corrugations. When they are cast off they leave behind more or less extensive **ulcers**, which are apt to coalesce, and thereby attain a very considerable size. Not infrequently the greater part of the mucous membrane is thus destroyed (Fig. 337 *a*), mere islands of the mucosa (*b*) persisting, and these, on account of the inflammatory swelling, generally tower above the ulcerated areas.

In diphtheritic inflammation the mucosa and submucosa are thickly infiltrated with cellular, serous, and in part fibrinous exudations (Fig. 336 *c*). Not infrequently they also contain patches of haemorrhagic extravasation. The muscular and serous coats are at times infiltrated with leucocytes.

Inflammation of the large intestine associated with ulceration is described generically as **dysentery** (Art. 202).

Dysenteric inflammation usually results from putrefactive decomposition of the intestinal contents, or from specific intestinal infection. It is however occasionally due to poisons, such as arsenic or corrosive sublimate, and to septicaemic conditions. The severe haemorrhagic and necrotic inflammation of the large intestine in cases of poisoning by corrosive sublimate is preceded by engorgement and thrombosis of the vessels of the mucosa.

Croupous inflammation, characterised by the presence of coagulated masses in the form of small flakes or large false membranes, is not uncommonly associated with catarrhal and diphtheritic inflammation. Thus, for example, the inflammation in dysentery may in the large intestine be mainly diphtheritic in character, while in the small intestine it is for the most part catarrhal and croupous. In some cases the diphtheritic sloughs are covered over with fibrinous false membranes.

Haemorrhages are very common in all forms of intestinal inflammation, the extravasation taking place chiefly from the ridges of the mucous folds. The seats of diphtheritic sloughing are frequently infiltrated with extravasated blood, and blood often mingles with the surface exudations.

The solitary and agminated **lymphadenoid follicles** of the intestine seem in many cases of enteritis to be practically unaffected, and are accordingly often discerned with difficulty or not at all. In other cases they are swollen, and project above the surface as whitish or greyish-white nodes, occasionally surrounded by a reddened areola. Peyer's patches when they swell assume the form both of nodes and of reticular elevations enclosing little pits. When the swelling is considerable, the entire patch is raised like a plateau above the general level, its surface being smooth or irregularly corrugated. All these conditions are included in the term **follicular enteritis**.

The swelling of the follicles may be due to mere hypertrophy (Fig. 338 *g*); indeed, it is often difficult or impossible to decide how far the enlargement is simply physiological, the lymphadenoid tissue of the intestine being normally subject to great variation, and how far it is morbid and due to infective or toxic influences. Follicular enteritis is most commonly met with in children (Fig. 338), and particularly in children who have died from diphtheria. The germinal centre of the follicle is in these cases enlarged, and the number of lymphoid cells migrating from the follicle to the surface is increased (Fig. 338 *h i*).

In many cases the swelling of the solitary follicles and agminate glands is mainly due to an excessive accumulation of lymphoid cells in the adjacent lymph-channels, and to circumfollicular or interfollicular cellular infiltration (Fig. 339 *h*). The corrugations and reticulations on the surface of Peyer's patches, and their general enlargement, are in great measure referable to these changes, which are most frequent in cases of infective or toxaemic catarrh. The follicles of the swollen patches are sometimes unchanged, sometimes swollen or degenerate, and in diphtheria they often enclose necrotic foci (*f*).

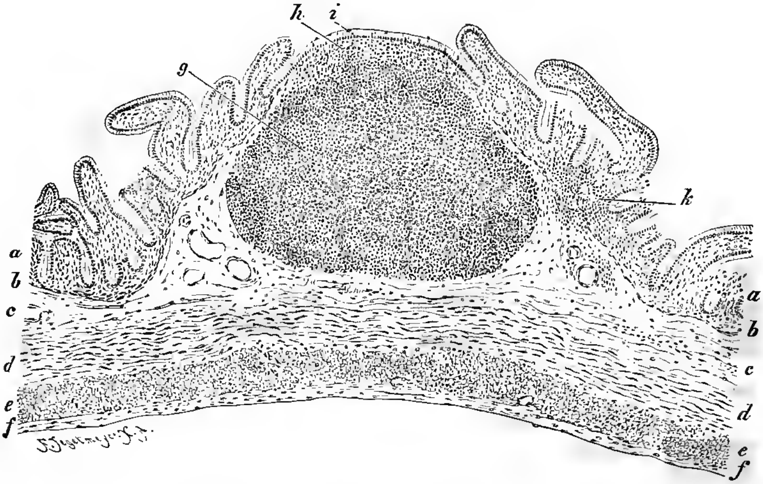

FIG. 338. SECTION THROUGH A HYPERTROPHIC FOLLICLE OF THE SMALL INTESTINE FROM A CHILD WHO DIED OF DIPHTHERIA.

(*Preparation hardened in Müller's fluid, stained with haematoxylin, and mounted in Canada balsam:* × 30)

a	glandular layer of the mucosa
b	*muscularis mucosae*
c	submucosa
d	inner, *e* outer muscular layer
f	serosa
g	follicle
h	connective tissue overlying the follicle and infiltrated with leucocytes
i	epithelium infiltrated with leucocytes
k	aggregation of small cells in the mucosa

The swelling of the follicles and the circumfollicular infiltration may undergo resolution, the cells decreasing in number and the infiltration so disappearing. Occasionally, however, suppuration of the follicular and circumfollicular tissue takes place, and **follicular abscesses** and ulcers are produced, whose form and site correspond to those of the original follicle. The ulcers appear as flask-shaped excavations reaching the submucosa, with overhanging borders and a relatively narrow opening to the surface. When the suppuration extends and neighbouring excavations run together, large sinuous ulcerations are produced.

The **lymph-vessels** of the intestinal wall take a more or less active part in the inflammation, often containing large numbers of leucocytes, and at times fibrin also. Desquamated endothelial cells, especially in the region of the muscular and serous coats, are frequently found within the lymph-vessels; and where the inflammation is of some standing they are filled with large epithelioid cells, some of them multinuclear, which are no doubt derived by proliferation from the endothelium.

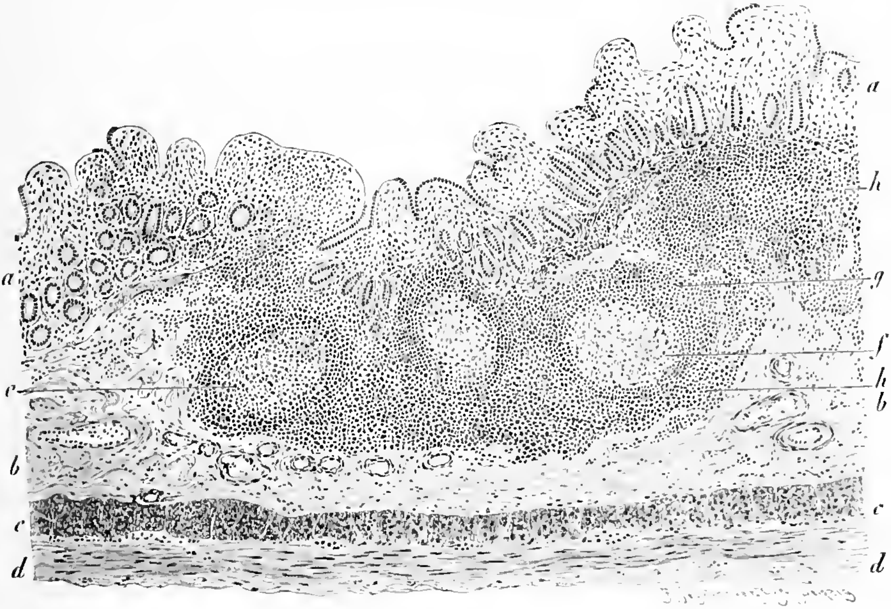

FIG. 339. BORDER OF A SWOLLEN PEYER'S PATCH FROM A CHILD WHO DIED OF DIPHTHERIA.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin* : ×45)

a b c d the several coats of the intestine
e normal follicle
f follicle with partially necrotic germinal centre
g h circum- and inter-follicular cellular infiltration of the submucosa

200. The slighter varieties of catarrh end in **recovery** without leaving any permanent change: the exudation is absorbed, and the lost epithelium is replaced by proliferous multiplication of the epithelial cells of the crypts of Lieberkühn. In catarrh of long standing the restoration of the tissue may not be complete, the outcome being a form of atrophy of the mucous membrane characterised by thinning of the glandular layer. This is of course more

marked in cases where the inflammation has resulted in suppuration and diphtheritic sloughing, with destruction of the whole thickness of the mucous membrane.

Atrophy of some part of the intestinal mucous membrane (Fig. 341) is thus very frequently discovered on post-mortem examination. Proliferous regeneration however always takes place (Fig. 340 *h i k*) at the denuded places, and leads to restoration not only of the epithelial surface, but of the fibrous and glandular structures also.

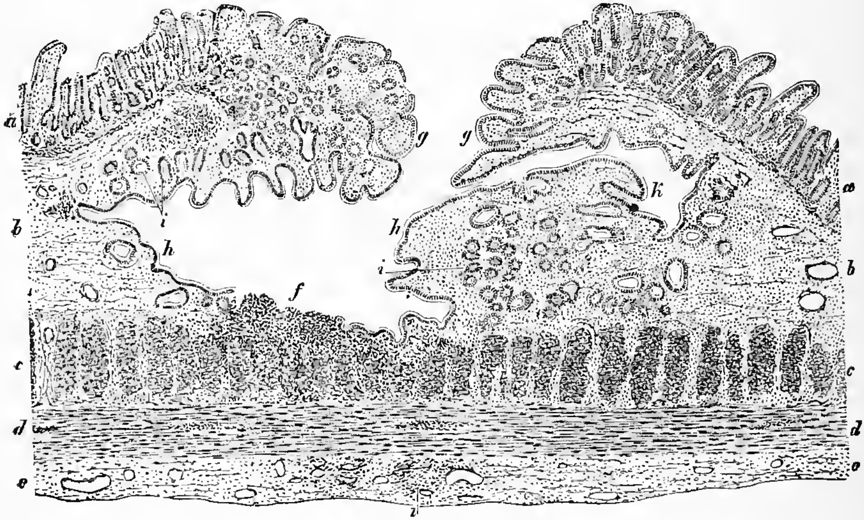

FIG. 340. HEALING OF AN ULCER OF THE SMALL INTESTINE WITH FORMATION OF GLANDULAR TUBULES IN THE SUBMUCOSA.

(*Preparation hardened in Müller's fluid, alcohol, and celloidin, stained with haematoxylin, and mounted in Canada balsam:* × 20)

a	mucosa
b	submucosa
c d	*muscularis mucosae*
e	serosa
f	part of the floor of the ulcer still uncovered with epithelium
g	overhanging margins of the ulcer
h	part of the floor covered over with epithelium
i	newly-formed gland-tubules situated in the submucosa
k	deep sinus lined with epithelium

Ulcers reaching even to the submucosa (Fig. 340 *f*) may become covered over with epithelium (*h k*), the adjacent submucosa becoming permeated by newly-formed glands (*i*); and thus, the floor being raised by the growth in it of proliferous fibrous tissue, the ulcerous excavation is ultimately filled up. But the regenerative process often remains incomplete, even when the glandular layer only has been affected.

Thus a more or less marked atrophy of certain portions of the intestinal mucous membrane occurs in the majority of adult cases subjected to post-mortem examination (according to NOTHNAGEL, in 80 per cent.). It is generally met with in the large intestine, and here most commonly about the caecum and ascending colon, less often in other portions of the large intestine and in the ileum. Not infrequently the glandular layer in the affected spots is entirely absent (Fig. 341 *e*), and in other parts the glands are more or less dwarfed and stunted (*a b*).

When the mucosa over large and continuous areas is entirely destroyed by ulceration, no reproduction of the glandular structures is possible. In such cases cicatrisation takes place by the formation of new connective tissue, derived either from the mucosa or from the submucosa, the latter often undergoing marked inflammatory thickening and induration. Such cicatricial hyperplasia, involving it may be the muscular layers also, sometimes encircles the bowel or the larger portion of its circumference, and by contraction gives rise to more or less grave intestinal **stricture**.

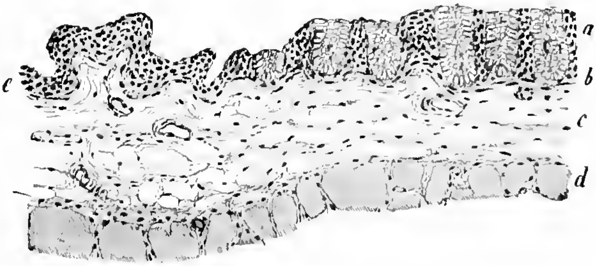

FIG. 341. MUCOSA AND SUBMUCOSA OF AN ATROPHIC PORTION OF THE LARGE INTESTINE.

(*Preparation hardened in Müller's fluid and alcohol, stained with alum-carmine, and mounted in Canada balsam:* × 80)

a glandular layer reduced to one-half of its normal thickness
b *muscularis mucosae*
c submucosa
d outer muscular coat
e completely atrophied mucosa

The inner surface of the intestine thus almost or wholly deprived of its glandular layer may appear perfectly smooth (Fig. 342 *a*). Not uncommonly however the smooth surface is interrupted by ridges and polypous excrescences (Fig. 342 *b c*). These are due simply to persistent remnants of mucous membrane of which the glandular elements, and often the connective tissue as well, have undergone proliferous overgrowth.

Frequently they contain not only dilated and convoluted tubules, but also branching glandular canals, enclosing **cysts** produced by retained secretion. Such cysts are also at times due to the accumulation of secretion within cavities formed from deep ulcers (Fig. 340 *f*) that have become lined with epithelium and new-formed mucus-secreting glands.

The polypous elevations of the glandular and cystic islands of mucous membrane are the result partly of the lowering of the general level about them, partly of the intrinsic enlargement of the remnants themselves; the more prominent portions of the polypi are however apt to be dragged upon by the onward passage of the intestinal contents and the peristalsis of the bowel, whereby in some cases they become elongated and ultimately pedunculated.

A very rare phenomenon is the formation of **gas-containing cysts** (WINANDS: Gaseous cysts in the intestinal wall and in perityphlitic false-membranes *Ziegler's Beiträge* XVII **1895**) in the course of chronic inflammations of the intestine. They occur in the intestinal wall or in false-membranes due to peritonitis, and appear as dilatations of the lymph-spaces. The cause of the condition and the mode of development of the gas contained in the cysts is still unexplained.

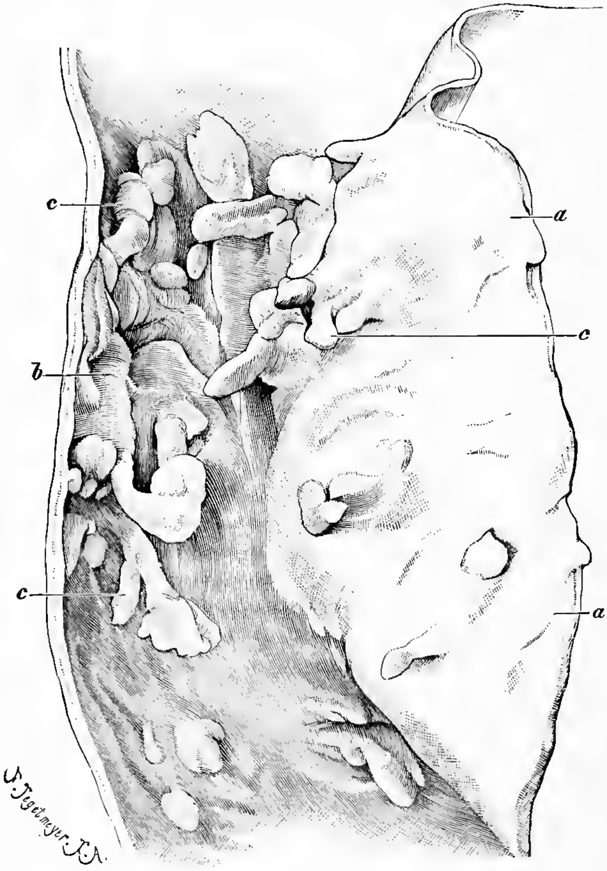

FIG. 342. POLYPOSIS OF THE LARGE INTESTINE FOLLOWING CHRONIC DYSENTERY.

a smooth and atrophic mucous membrane devoid of glands, with a few polypous growths

b mucous membrane containing glands

c mucous polypi

References on the Morbid Anatomy and Aetiology of Intestinal Inflammations (see also Arts. 201 and 202).

Baginsky: Intestinal catarrh *D. med. Woch.* **1885**; Putrefaction in the intestinal canal of children *ibidem* **1888**; Cholera infantum *A. f. Kinderheilk.* XII **1890**

Bertrand and Fontan: *De l'entéro-colite chronique endémique des pays chauds* Paris **1887**

Damaschino: *Maladies des voies digestives* Paris **1880**

von Davidoff: The relation of the intestinal epithelium to lymphadenoid tissue *A. f. mikrosk. Anat.* XXIX **1887**

Escherich: *Die Darmbakterien des Säuglings* Stuttgart **1886**; The bacteria of the intestine with special reference to those of infancy *Cent. f. Bakteriologie* I **1887**; Putrefaction in the intestinal canal of children *D. med. Woch.* **1888**

Fischer: Affections of the bowels in nephritis and uraemia *V. A.* 134 **1893** (with references)

Fraenkel, E.: Cysts of the intestinal canal *V. A.* 87 **1882**

Gairdner and Coats: *Lectures to practitioners on the diseases classified by the Registrar-general as Tabes mesenterica* London **1888**

Grawitz: Inflammation of the large intestine in mercurial poisoning *D. med. Woch.* **1888**

Heidenhain: The mucous membrane of the small intestine *Pflüger's Arch.* 43 **1889**

Kaufmann: *Die Sublimatintoxication* Berlin **1888**, and *V. A.* 117 **1889**

Kitagawa: Enteritis membranacea *Z. f. klin. Med.* 18 **1890**

Krysinski: *Ueber Enteritis membranacea* Jena **1884**

Kunkel: Acute mercurial poisoning *Sitzungsber. d. phys.-med. Gesellsch. zu Würzburg* **1889**

Lesage: Dyspepsia and green diarrhoea of infancy *Rev. de méd.* VII **1887**; The bacillus of the green diarrhoea of infancy *A. de physiol.* I **1888**

Nothnagel: *Beiträge zur Physiol. und Pathol. des Darmes* Berlin **1884**

Passow: Quantitative relations of the solitary follicles and lymphadenoid patches in the small intestine *V. A.* 101 **1885**

Rosenheim: *Art.* Inflammations of the intestine *Eulenburg's encyklop. Jahrb.* IV **1894**

Schwarck: Croupous and diphtheritic inflammations of the intestine *Inaug. Diss.* Bonn **1880**

Stöhr: Lymph-follicles of the intestine *A. f. mikrosk. Anat.* XXXVIII **1889**

Virchow: Abdominal affections *V. A.* 5 **1853**; Typhoid and dysentery of armies *V. A.* 32 **1871**

Woodward: *The med. and surgical history of the War of the Rebellion* (part II vol. 1 *Medical history*) Philadelphia **1879**

201. Inflammations of the bowel have received various names according to the particular parts affected. Several of these local inflammations exhibit peculiarities depending upon the anatomical relations of their respective seats. The chief of them are the following.

(1) **Duodenitis**, or inflammation of the duodenum, is usually associated with inflammation of the stomach. It not infrequently leads to obstruction at the mouth of the bile-duct, and thereby to retention of bile and jaundice (**catarrhal jaundice**).

The duodenum is also the seat of simple or perforating ulcer, corresponding to perforating ulcer of the stomach, and like it dependent on digestive corrosion.

(2) **Ileitis,** inflammation of the ileum, is often marked by the swelling and prominence of the solitary and agminate follicles.

(3) **Typhlitis** and **perityphlitis** imply inflammation of the caecum and vermiform appendage and the parts around them.

The vermiform appendage is peculiarly adapted to catch and retain substances passing through the caecum. Matters which have been swallowed, such as grape-seeds, apple-pips, cherry-stones, and the like, and faeces containing bacteria, may accumulate in the appendage and set up inflammation. Sometimes these become crusted over with phosphates and carbonates and so form faecal concretions or calculi.

The inflammation thus set up (clinically described as **appendicitis**) may extend to all the coats of the appendage and then attack the contiguous structures, and in this way necrosis and gangrene of the different coats with perforation may be caused.

The issue is comparatively favourable if the inflammation continues to be circumscribed while the exudation is moderate in amount, protective adhesions and false membranes being formed about the affected spot. It is much more unfavourable when perforation takes place before adequate adhesions are formed; fatal peritonitis is then nearly always induced. When perforation takes place into a part of the peritoneum shut off by adhesions, a sacculated faecal abscess is produced, which may burst internally or externally. Sometimes the appendage is entirely obliterated by adhesive inflammation; but if the inner or intestinal end becomes closed while the remainder continues to be patent, the natural mucous secretion may collect in the latter and distend it into a **cyst.**

Typhlitis and perityphlitis are sometimes due to the extension by continuity of inflammation already existing in more distant parts of the caecum or colon. Tuberculous and typhoid ulceration confined to the vermiform appendage may give rise to the formation of ulcers.

Obliteration of the vermiform appendage, which proceeds from the peripheral end inwards, may take place either with or without inflammation, and in old age is a very frequent phenomenon (RIBBERT).

(4) **Colitis,** or inflammation of the large intestine, is sometimes due to stoppage and accumulation of faecal masses or **scybala,** sometimes to septic infection, sometimes to the action of a specific poison.

(5) **Proctitis** is an inflammation of the rectum. In many points it resembles inflammation of the vermiform appendage. Foreign matters and stagnating faeces are frequently the exciting agents; but disturbances of the circulation in the haemorrhoidal veins, and traumatic injuries, such as are sometimes produced in

administering enemata, may likewise end by inducing inflammatory change in this part of the bowel.

Proctitis often results in the formation of ulcers and of fibrous hyperplasia, the latter taking the form of induration of the mucosa and submucosa or of polypous excrescences. The inflamed surface is usually covered with a muco-purulent exudation. When the inflammation and ulceration extend deeply into the tissues of the wall of the bowel, the surrounding connective tissue becomes infiltrated and hyperplastic, or breaks down into abscesses containing foetid pus (**periproctitis**). The ulcers of the mucosa and submucosa come in this way to communicate with burrowing sinuses and fistulous tracks covered with epithelium and extending into the surrounding parts, which are called incomplete or blind **internal fistulae.** Circumscribed or enclosed periproctal abscesses may break outwardly, and then constitute incomplete or blind **external fistulae.** Fistulae which communicate both with the rectum and with the exterior are called complete rectal fistulae or **fistulae in ano.** Fistulae occasionally communicate with the bladder or vagina.

Specific poisons like those of syphilis, tuberculosis, or dysentery, and cancer in the ulcerative stage, are liable to give rise to morbid changes of a similar kind. There may even be a primary periproctitis without any antecedent rectal ulceration, especially in connexion with pyaemia, typhoid, acute articular rheumatism, and puerperal septicaemia.

References on Typhlitis, Perityphlitis, and Proctitis.

Bierhoff: *D. A. f. klin. Med.* XXXVII **1886**
Cornil: *A. de physiol.* III **1873**
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Esmarch: Diseases of the rectum and anus *Deutsche Chirurgie* part 48
Graser: Peritonitis from the vermiform appendage *A. f. klin. Chir.* XLI **1891**
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Kelynack: *Pathology of the vermiform appendix* London **1894**
Nickel: Syphilitic ulcers of the rectum *V. A.* 127 **1892**
Nordmann: Lesions of the rectum from enemata *Inaug. Diss.* Basle **1887**
Paltauf: Spontaneous rupture of the large intestine in the new-born infant *V. A.* 111 **1888**
Poelchen: Aetiology of ulcers of the rectum producing stricture *V. A.* 127 **1892**
Quénu: Haemorrhoids etc. *Rev. de chir.* **1892**
Ribbert: Morbid anatomy of the vermiform appendage *V. A.* 132 **1893**
Sonnenburg: Perityphlitis *D. Z. f. Chir.* 38 **1894**
Steiner: Morbid anatomy of the vermiform appendage *Inaug. Diss.* Basle **1882**

202. **Dysentery** is an epidemic or sporadic inflammatory affection of the large intestine, due to the action of a specific virus. The exact nature of the virus of epidemic dysentery is unknown; it is however probable that all epidemics are not caused by the

same virus, in other words that under the term dysentery are included several intestinal affections that are aetiologically distinct. Thus the dysentery of Egypt is not of the same nature as the variety that is occasionally epidemic in various parts of Europe, and this again is different from the forms that occur sporadically. In some cases abnormal decomposition of the intestinal contents, poisoning by corrosive sublimate and calomel, and septic infections, give rise to inflammatory processes whose clinical course resembles that of dysentery.

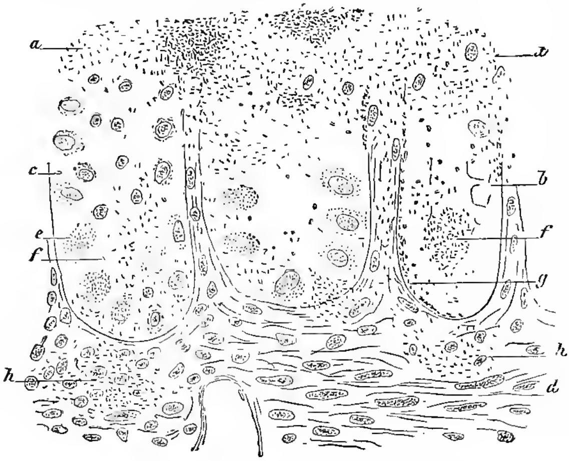

Fig. 343. Acute dysentery or bacillary diphtheritis of the large intestine.

(*Preparation hardened in alcohol, stained with gentian-violet, and mounted in Canada balsam:* × 300)

a	necrotic tissue containing the bacilli
b	gland with necrotic epithelium
c	gland with desquamated epithelium
d	connective tissue
e	degenerate desquamated epithelial cells
f	bacilli in the interior of the gland
g	layer of bacilli just beneath the epithelium
h	colony of bacilli in the connective tissue

Some at least of the epidemic forms are due to bacterial infection: among the epidemics that have occurred in Europe certain have been so intimately associated with bacterial invasion of the intestine (Fig. 343 *f g h* and Fig. 344 *e*) that the causal connexion of the micro-organisms with the disease can hardly be doubted. The micro-organisms in question are minute bacilli (Fig. 343 *f g h*), and they lie scattered (*a*) or aggregated (*f g h*) within the glands (*f*), the glandular epithelium (*g*), and the connective tissue (*h*). Their multiplication in the tissues is followed by inflammation (Fig. 344 *h*), necrosis (Fig. 343 *a b* and Fig. 344 *a g*), and degeneration (Fig. 343 *e*). The observations of Kartulis, Kruse, Pasquale, Osler, Roos, and others have rendered it probable that a variety of dysentery exists which is caused by **amoebae**, and that this amoebic dysentery occurs chiefly in Egypt and Greece, though it is also met with in other countries, such as Russia, Germany, North America, etc.

The intensity and extent of the dysenteric inflammation vary in different cases. It may be restricted to the rectum, sigmoid flexure, and descending colon, or it may reach up to or a little beyond the ileo-caecal valve. Often too in the same case the various parts of the tract are variously affected.

In recent cases the mucous membrane is highly congested and swollen, and generally beset with minute extravasations of blood. The epithelial surface is overlaid with glairy blood-streaked mucus. This presently becomes more puriform and blood-stained, and interspersed with the flaky fibrinous shreds and films (described in Art. 199) which indicate the beginning of superficial necrosis of the mucous membrane. Soon the necrosis is made sufficiently evident by the appearance of erosions and losses of substance.

We might perhaps distinguish a catarrhal and a diphtheritic form of dysenteric inflammation, but in practice the one passes insensibly into the other and the distinction is inappreciable. In slighter cases the necrosis and loss of substance are at first merely superficial (Fig. 336 *f*); but the deeper structures are successively attacked (*g*), and in severe cases the greater part or the whole of the glandular layer of the mucous membrane at particular spots perishes (Fig. 344 *a*). The necrotic tissue is reduced to a turbid granular mass, in which the structural elements and the nuclei of the cells soon cease to be recognisable. The parts which undergo necrosis are generally confined to the prominent ridges and folds of the mucous membrane; these appear dirty-grey or black, while the intervening parts are still livid or dark-red. In other cases the necrotic tissue takes the form of a more or less adherent flaky coating, or more rarely of broad continuous sloughs. The underlying tissue is in all cases densely infiltrated

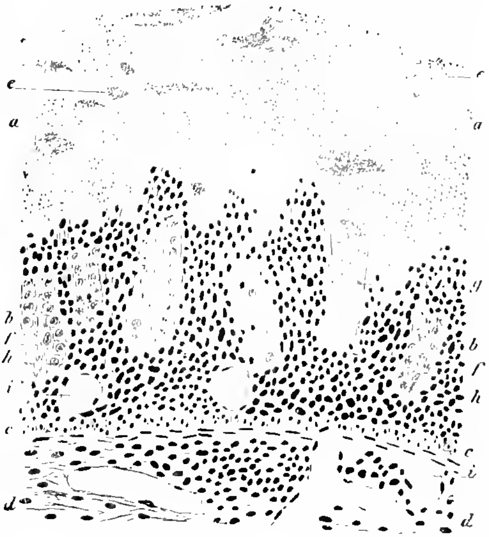

FIG. 344. BACILLARY DIPHTHERITIS (DYSENTERY) OF THE LARGE INTESTINE.

(*Preparation hardened in alcohol, stained with gentian-violet, and mounted in Canada balsam*: × 80)

a	necrotic portion of the glandular layer of the mucosa beset with bacilli
b	unaffected glands
c	*muscularis mucosae*
d	submucosa
e	colonies of bacilli
f	glands showing normal epithelial cells
g	glands with necrotic epithelial cells and bacilli
h	connective tissue infiltrated with cells
i	blood-vessels

with cells (Fig. 336 d_1 and Fig. 344 *h*). The infiltration occasionally extends through the entire thickness of the submucosa (Fig. 336 *e*), and at length invades the muscular layers. The lymphadenoid follicles also take part in the process, and frequently ulcerate. Occasionally a portion of the mucosa is undermined by ulceration beneath it, and in this way broad patches of the tissue are separated and cast off.

When portions of the mucosa are thus removed, open ulcers are of course left behind. These vary much in their depth and extent: sometimes over a great part of the bowel the mucous membrane persists only in narrow strips and islands. Amoebic dysentery is said to be characterised by the formation of small circumscribed ulcers with undermined edges.

The affection may come to a standstill at various stages of its course, and repair then begins. The slighter cases, in which but little substance is lost, are naturally the readiest to heal; but a certain amount of atrophy of the mucosa always remains. When the ulcerative process has gone further atrophic cicatrices are left to mark the site of the injury. In severe cases accompanied by great destruction of tissue, in which the acute specific process is succeeded by chronic inflammation, the whole structure of the bowel is altered in a remarkable way. Over broad areas the glandular layer is almost or altogether absent (Fig. 337 *a*); the deeper layers of the mucosa and submucosa look tough and indurated; the connective structures are hyperplastic; and the other coats are likewise dense, thickened, and unyielding. The channel of the intestine is usually narrowed, often so much so that a finger can hardly be introduced. The mucous membrane is only recognisable in isolated patches here and there, and these not infrequently assume the form of papillary or polypous outgrowths (Fig. 342) from the general surface. Small cysts lined with cylindrical epithelium are frequently formed by dilatation of glandular tubules which have become abstricted and occluded, and by the accumulation of secretion in ulcerous cavities that have become covered over with epithelium. This condition is usually accompanied by abundant muco-purulent discharge from the diseased surfaces, and constitutes what is clinically described as chronic dysentery or **coeliac flux**.

References on Dysentery.

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Bertrand and Fontan: *De l'entéro-colite chronique endémique des pays chauds* Paris **1887**

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Councilman and Lafleur: Amoebic dysentery *Johns Hopkins Hosp. Rep.* II Baltimore **1891**

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KELSCH: Chronic dysentery *A. de physiol.* V **1873**; (with KIENER) *A. de physiol.* III **1884**
KRUSE and PASQUALE: Dysentery and abscess of the liver *Z. f. Hygiene* XVI **1894**
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OGATA: Aetiology of dysentery *Cent. f. Bakteriologie* XI **1892**
OSLER: Concerning the amoeba of dysentery *Cent. f. Bakteriologie* VII **1890**
ROOS: Amoebic enteritis *A. f. exp. Path.* XXXIII **1894**
WESENER: Our present knowledge of dysentery *Cent. f. allg. Path.* III **1892**
WOODWARD: *The medical and surgical history of the War of the Rebellion* part II Philadelphia **1879**

203. **Epidemic** or **Asiatic cholera** is characterised anatomically by the presence of an acute inflammation extending over the whole of the intestine, accompanied by an enormously copious transudation of liquid through the mucous membrane. In cases which prove fatal in the first two or three days, the bowel is found to contain a great quantity of a turbid, greyish, inodorous alkaline liquid, often mingled with minute shreds and flakes of mucus—the so-called "rice-water" stools. The mucous membrane is moist, pink, injected, and swollen, and here and there beset with haemorrhagic spots. Usually the serous surface is injected and turbid, and feels sticky to the touch. The epithelium of the more superficial portions of the crypts within the first few hours of the disease undergoes mucoid degeneration and desquamation, and the surface of the intestine becomes thereby covered with slime. Later on most of the epithelium exfoliates and mingles with the transuded liquid. The connective tissue of the mucosa, and to some extent of the submucosa also, is more or less thickly infiltrated with leucocytes, which sometimes penetrate even to the serous coat. The follicles of the small intestine are somewhat swollen, greyish-white or bright pink in colour, and surrounded by a hyperaemic areola. The ileum is usually the part most affected, the large intestine being often almost unaltered, at least in the early stages.

When death does not occur until a later stage of the disease, the appearance of the intestine is notably different. The contents are scanty and less liquid, and at the same time they show more signs of the presence of bile: in the large intestine scybalous masses are sometimes found. The mucous membrane is pale or slate-coloured, or it may be injected and beset with minute haemorrhages. Ulcers make their appearance in the stage of collapse, especially in the colon and lower part of the ileum, as the result of diphtheritic necrosis and sloughing. Sometimes the large intestine has almost the same look as in dysentery.

The specific virus of cholera is the **spirillum** (or comma-bacillus) discovered by Koch. In recent cases large numbers of these microbes are found in the intestinal contents and in the layers of epithelium that are undergoing mucoid change and desquamation. They are also found in the lumina of the crypts, between and beneath their epithelium, and in the subepithelial connective tissue. The spirilla as they multiply produce a chemical poison or toxin, which acts injuriously upon the intestinal epithelium, and when absorbed affects the entire organism, in particular giving rise to vaso-motor paralysis. The acid gastric juice of a healthy person checks the development of the spirilla; and thus disorder of the gastric functions predisposes to choleraic infection.

Cholera nostras or European cholera leads to morbid changes in the intestine similar to those seen in Asiatic cholera; but its aetiology is different. In a given case however the diagnosis can only be made, apart from considerations as to the general course of the epidemic, by the unmistakable demonstration of the spirilla characteristic of the Asiatic disease. These multiply in a peculiar manner on artificial cultivation, produce certain definite changes in the culture-media, and induce specific effects when injected into animals. The bacteriology of the subject is dealt with in the volume on General Pathological Anatomy.

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Wood: *Reports Lab. Roy. Coll. Physicians* II Edinburgh **1890**

204. **Typhoid** (or enteric) **fever** is an infective disease, due to the growth and multiplication of the typhoid bacillus in the intestine.

The morbid changes in typhoid appear chiefly in the lower part of the ileum and the upper part of the colon; they are seldom met with much higher or much lower in the intestine. The changes consist essentially of a necrotic inflammatory infiltration of the follicular structures and the parts around them, accompanied by a catarrhal inflammation of the rest of the mucous membrane.

In the first few days of the attack the mucous membrane of the lower part of the ileum and its agminate glands or Peyer's patches are intensely congested and uniformly swollen. Soon the swelling of the patches becomes more marked, raised and winding ridges appearing on their surface (Fig. 345).

The swelling extends more or less quickly over the whole of each patch, so that it has in general the look of a flat elevation or plateau rising above the general surface. When the swelling is at its height the ridges generally become levelled up as it were, and are no more distinguishable: the surface of the patch is then smooth or pitted with minute depressions corresponding to the sites of the individual follicles. The solitary follicles take the form of rounded nodules as a result of the same process (Fig. 345).

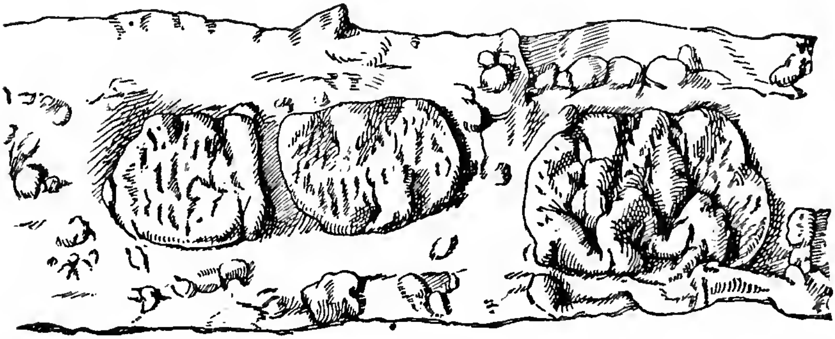

Fig. 345. ENLARGEMENT OF THE SOLITARY FOLLICLES AND OF THE PEYER'S PATCHES IN TYPHOID FEVER.

(Reduced to four-fifths of the natural size)

When this stage of swelling is complete the patches and follicles, which at first were bright-red in colour, become pale and creamy-white.

The swelling of the patches and follicles is chiefly due to the extreme cellular infiltration of the mucosa (Fig. 346 a_1), and submucosa (b_1), from extravasation and proliferation of the follicular and the circum- and inter-follicular tissue. The glands (f) of the mucosa are thereby thrust asunder and displaced, and the villi are likewise infiltrated and swollen. The submucosa underlying the patches is uniformly infiltrated (b_1). In the earlier stages the several follicles (g) within the infiltrated region are still distinct and recognisable, but later on they are no longer to be separately distinguished. The muscular coat ($c_1 d_1$) and the serosa (e_1) are sometimes, though to a minor degree, involved in the cellular infiltration and proliferation.

The number of swollen patches and follicles varies much. Often but a small number or even a single one is markedly affected, while in other cases the disease extends upwards to the jejunum or downwards to the anus.

In the second week of the disease partial disintegration and necrosis of the swollen patches usually set in (Fig. 347). The disintegration affects the whole of the central part of the patch (*a*), or several parts of it simultaneously. The surface quickly assumes a frayed or ragged appearance, and becomes yellow or brown from imbibition of bile. According to MARCHAND croupous or fibrinous deposits are occasionally formed on the surface of the swollen patches; these deposits are adherent, and so have the appearance of undetached sloughs. Gradually the disintegrated and necrotic tissue becomes loosened from its attachments to the surviving structures, and erosion of the vessels leading to haemorrhage is then apt to take place.

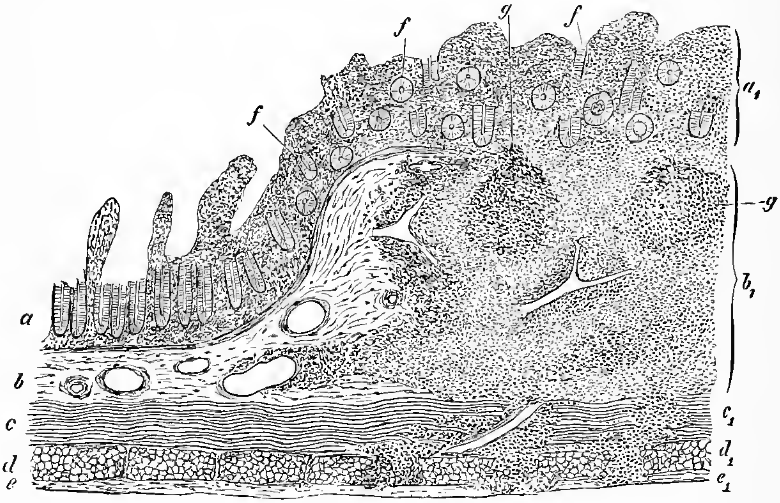

FIG. 346. SECTION THROUGH THE MARGIN OF A PEYER'S PATCH FROM A CASE OF TYPHOID FEVER.

(Preparation hardened in alcohol, stained with Bismarck-brown, and mounted in Canada balsam: × 15)

a	mucosa	a_1 b_1 c_1 d_1 e_1	the same layers infiltrated and swollen
b	submucosa	*f*	crypts cut through transversely
c	internal muscular coat	*g*	lymphadenoid follicles
d	external muscular coat		
e	serous coat		

After the separation of the sloughs, which takes place within a few days, an erosion or **typhoid ulcer** (*b c*) is left, the floor of which generally looks smooth and clean. The borders of the ulcer are at this stage still swollen and infiltrated.

The ulcers usually remain co-extensive with or very slightly overpass the area of the infiltrated Peyer's patches and follicles; they rarely invade the tissue beyond. Cases however occur in which, especially around the ileo-caecal valve, extensive tracts of mucous membrane are attacked and disintegrated by the advance of the ulcerative process. Necrosis seldom goes beyond the mucosa and submucosa, though the muscular and finally the serous coat may be affected, but the necrosis here is never so extensive as in the mucosa and submucosa.

When the inflammation and necrosis involve the external layers, **perforation** of the intestine and peritonitis is apt to ensue.

The processes of absorption and repair begin at various stages of the disease. If no necrosis takes place, the swelling of the patches goes down as the infiltrated material is absorbed: the patches thereupon become less turgid, and once more hyperaemic. When red corpuscles escape from the damaged vessels the patches take on a red or blood-stained tint which presently turns to a slaty-grey.

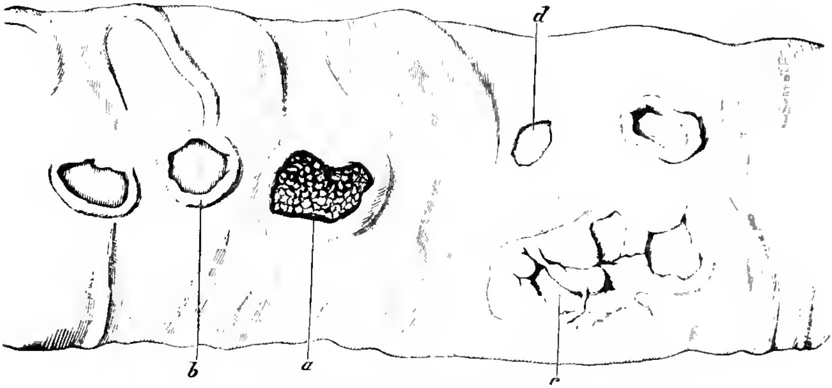

FIG. 347. INTESTINAL ULCERS IN TYPHOID FEVER.

(*Natural size*)

- *a* ulcer with adherent necrotic slough
- *b* clean ulcer with infiltrated border
- *c* Peyer's patch containing several ulcers with swollen edges
- *d* ulcers whose edges are no longer swollen

The infiltrated borders of the typhoid ulcers become reduced and softened and hyperaemic by the same steps. Often enough considerable haemorrhage ensues, leading not only to haemorrhagic infiltration of the tissue, but to actual effusion of blood into the intestinal canal. As the healing process goes on, delicate granulation-tissue is formed in the floor of the ulcer, which soon receives an investment of epithelium. This at length is furnished with groups of new but irregularly-arranged glandular tubules, some of which are in direct contact with the muscular coat.

The site of a former typhoid ulcer appears for a long time after as a smooth shallow depression, slaty-grey in colour, within which the muscular coat is covered only with highly-cellular connective tissue, with or without glands, and a single layer of epithelial cells.

The typhoid bacilli are found during the first eight or ten days of the fever within the swollen patches. According to A. Pfeiffer, they are occasionally discoverable in the intestinal contents and in the stools.

The characteristic inflammation of the lymphadenoid structures of the intestine in typhoid is sometimes accompanied by an inflammation of the corresponding mesenteric glands, which is apt in like manner to induce partial necrosis in them.

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Cygnäus: Researches on the typhoid bacillus *Ziegler's Beiträge* vii **1890**
Hoffmann, C. E. E.: *Untersuchungen über die pathologisch-anatomischen Veränderungen der Organe bei Abdominaltyphus* Leipzig **1869**
Klein: Report on the intimate changes in enteric or typhoid fever *Reports of the Medical Officer of the Privy Council* London **1875**
Marchand: Intestinal changes in typhoid fever *Cent. f. allg. Path.* i **1890**
Virchow: Typhoid and dysentery in armies *V. A.* 52 **1871**
Zülzer: *Art.* Typhoid fever *Eulenburg's Realencyklop.*

205. **Tuberculosis** of the bowel is one of the commonest of intestinal diseases, and chiefly attacks the lymphadenoid structures. The neighbourhood of the ileo-caecal valve is the region most frequently affected, but often enough the whole of the large intestine down to the anus becomes tuberculous, and in the other direction the disease may extend to the duodenum.

At first a little nodule covered with epithelium protrudes from the surface of a Peyer's patch or over a solitary follicle. After a time the centre of the nodule becomes pale-yellow, indicating that necrosis and caseation have begun. The caseous parts break down and a **tuberculous ulcer** (Fig. 348 *h*) with infiltrated borders is formed. New tubercles are formed in the floor and margins (i i_1), and between them the tissue becomes infiltrated with cells. When the necrosis and caseation extend to these intervening parts, the ulcers expand and coalesce with others formed in a like manner (h_1).

Ulcers of any great size (Fig. 349 *b*) are usually very irregular in their outline. Some are rounded, but more are oval or at least elongated, the longer axis being transverse to the axis of the bowel: others again are sinuous and spreading.

The borders are in general infiltrated, but in the larger ulcers not uniformly so. The edges and the floor are here and there beset with yellow necrotic nodules.

The eruption of tubercles extends very frequently from the submucosa to the muscular coat (Fig. 348 *c d i*), and finally reaches the serous surface, following the course of the lymph-

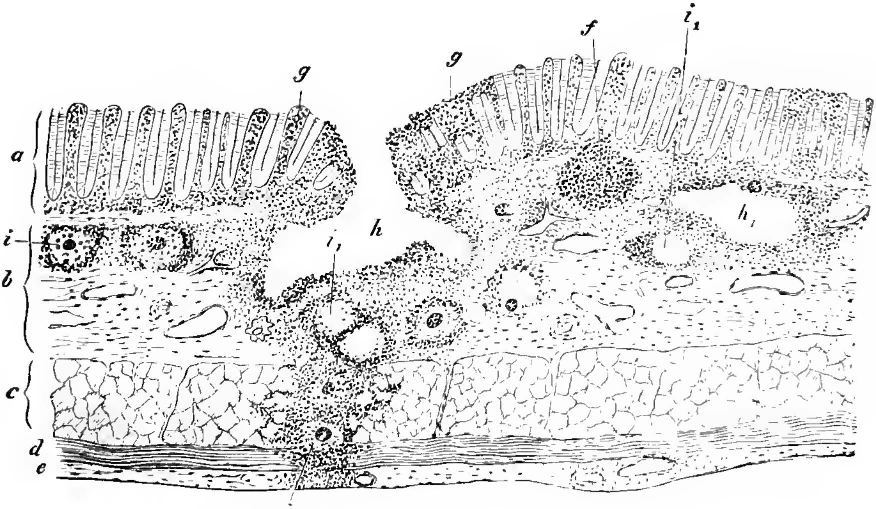

FIG. 348. TUBERCULOSIS OF THE LARGE INTESTINE.

(*Bismarck-brown staining:* × 30)

a	mucosa	*g*	cellular infiltration of the mucosa
b	submucosa	*h*	tuberculous ulcer
c	internal muscular coat	h_1	submucous abscess
d	external muscular coat	*i*	recent or grey tubercle
e	serous coat	i_1	caseous or yellow tubercle
f	solitary follicle		

channels. On the serous surface the tubercles appear in groups or beaded rows, the membrane in the neighbourhood being reddened by the injection of numerous blood-vessels, some of them newly formed.

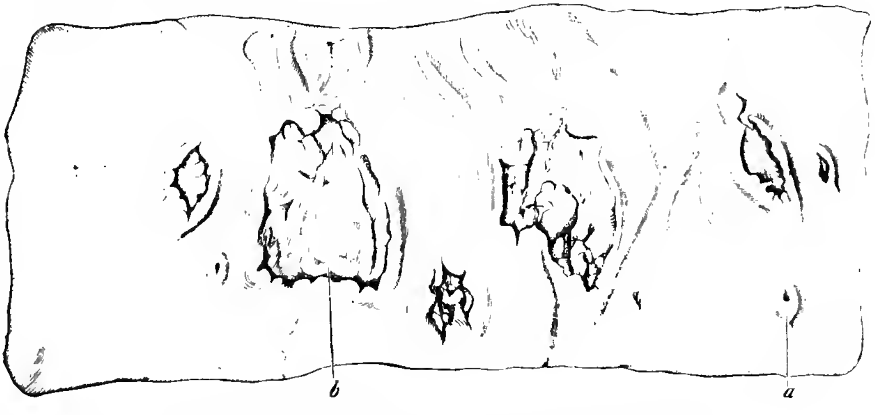

FIG. 349. TUBERCULOUS ULCERS OF THE INTESTINE.

a	small follicular ulcer	*b*	large ulcer

2 x

Only in rare cases does tuberculous ulceration come to a standstill and issue in more or less complete cicatrisation. As a rule it advances steadily, involving more and more of the bowel until the patient dies. When cicatricial contraction takes place in the floor of an encircling ulcer, or the eruption of tubercles and the consequent fibrous hyperplasia become excessive, and adhesions of the affected intestine with neighbouring parts are set up, the result is more or less severe stricture of the bowel, which may even pass into complete obstruction. If the ulceration extends to the outer layers of the intestinal wall, perforation may occur and give rise to purulent or putrid peritonitis. Ischiorectal abscesses not infrequently form in connexion with ulcerative tuberculosis of the rectum, and these often lead to the formation of external, internal, or even complete **fistulae**.

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Biedert: Tuberculosis of the intestine and of the lymphatic system *Jahrb. f. Kinderheilk.* XXI **1884**

Dobroklowsky: Penetration of the mucous membrane of the intestine by tubercle-bacilli *A. de méd. exp.* II **1890**

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Herxheimer: Bacilli in caseous intestinal follicles *D. med. Woch.* **1885**

Litten: Tuberculous stricture of the intestine *Z. f. klin. Med.* II **1881**

Spillmann: Tuberculosis of the intestinal tract *Thèse* Paris **1878**

Tchistowitch: Intestinal tuberculosis *Ann. de l'Inst. Pasteur* III Paris **1889**

Volkmann: Tuberculous fistulae of the anus *A. f. klin. Chir.* XXXII **1885**

206. **Syphilis** of the intestine, in the form of chancres or initial scleroses, moist papules, and gummata, occurs most commonly in the rectum immediately above the anus. Chancres are generally due to direct venereal infection, and when once formed lead quickly to the production of ulcers. Papules usually appear simultaneously with mucous patches about the anus, and they likewise pass into ulcers. Gummatous foci are found chiefly in the submucosa, and as they break down and rupture on the surface give rise to deep ulcerous excavations. The mucosa for 10 or 12 centimetres above the anus is sometimes so completely destroyed that only small round patches and strips of the membrane are left, and these are often undermined. So long as the disease is recent, the ulcers secrete pus; as they heal, the underlying tissue becomes indurated and contracts, leading to stricture of the rectum.

In rare instances gummatous nodes develope in the tissue surrounding the rectum. As these disintegrate and rupture into the bowel or towards the skin, they give rise to internal or external fistulae.

Syphilitic inflammations of the colon or small intestine are extremely rare; but ulcers, radiating cicatrices, and nodose or diffuse infiltrations of the intestinal wall that cannot be regarded as other than syphilitic are occasionally observed. These intestinal manifestations are met with chiefly in children suffering from inherited syphilis. They assume the form of diffuse mucous and submucous growths, situated within or between the Peyer's patches. Such granulomatous growths may break down into ulcers which sometimes encircle the bowel.

References on Syphilis of the Intestine.

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Eberth: Hereditary syphilis *V. A.* 40 **1867**
Förster: Hereditary syphilis *Würzburg. med. Zeit.* IV **1863**
Hayem and Tissier: Syphilis of the intestine *Rev. de méd.* IX **1889**
Homén: Multiple syphilitic ulcers in the small intestine *Cent. f. allg. Path.* **1893**
Huet: Syphilitic affections of the rectum *Behrend's Syphilidologie* new series II **1860**
Israel, O.: A rare variety of annular ulcer of the intestine *Charité-Annalen* IX **1884**
Jürgens: Intestinal syphilis *Berl. klin. Woch.* XVII **1880**
Lancereaux: *Traité histor. et prat. de la syphilis* Paris **1873**
Lang: *Vorles. über Path. und Therap. d. Syphilis* Wiesbaden **1885**
Mřaček: Hereditary syphilis *V. f. Derm. u. Syph.* X **1883**
Oser: Three cases of syphilitic enteritis *A. f. Derm. u. Syph.* III **1871**
Rieder: Intestinal affections due to acquired syphilis *Jahrb. d. Hamburger Krankenanstalt* I **1890**
Roth: Hereditary syphilis *V. A.* 43 **1868**
Schott: Hereditary syphilis *Jahrb. f. Kinderheilk.* **1861**
Schwimmer: *A. f. Derm. und Syph.* V **1873**
Virchow: *Die krankhaften Geschwülste* Berlin **1863**

207. **Intestinal anthrax** is an affection of the bowel due to the *Bacillus anthracis*, which is either introduced directly with the food (primary anthrax), or reaches the intestine by way of the blood from another point of infection (secondary anthrax).

Most commonly the disease is limited to the jejunum, duodenum, and ileum; it rarely appears in the stomach and large intestine. The affected parts somewhat resemble the malignant pustules of the skin, appearing as blackish-red or brownish-red haemorrhagic patches, varying in size from that of a lentil to that of a bean, with a greyish-yellow or greenish-yellow dirty-looking slough in the centre. In other cases the ridges of the intestinal folds are swollen and infiltrated with extravasated blood, while the most prominent portions are sloughy. The mucosa and submucosa within the affected area are haemorrhagic, and the tissue around it is oedematous and hyperaemic. Bacilli abound in the patches and in the surrounding tissue, especially in the blood-vessels and lymphatics and in the swollen lymph-glands corresponding to the diseased parts.

Intestinal anthrax is often referred to as **intestinal mycosis** (*enteromycosis bacteritica*). This latter term, however, is a general one, and is applicable to other mycotic affections of the bowel that have no relation to anthrax. Cases of meat-poisoning, a group of septicaemic affections due to the ingestion of meat from animals that have died of septic disease, or of flesh in a state of incipient putrefaction, are regarded as examples of intestinal mycosis. The morbid changes in the bowel vary from simple catarrh to croupous and diphtheritic inflammation, or to lesions resembling those of anthrax or typhoid fever: the symptoms are those of virulent poisoning, and are probably due to putrefactive alkaloids or ptomaines. The production of the ptomaines may either precede or follow the ingestion of the unwholesome meat. In the latter case the multiplication of putrefactive bacteria within the intestine is usually the cause. These in some cases settle and produce colonies in the intestinal wall itself, and so induce local changes in it.

Actinomycosis, when it starts in the intestine, begins by forming nodular granulomatous deposits in the mucosa and submucosa: these contain the specific fungi, and presently break down and ulcerate. The process may extend to the peritoneum, the retroperitoneal tissue, or to the adjacent organs. It sometimes causes perforation of the bowel, resulting in faecal abscess and peritonitis.

References on Anthrax and Enteromycosis.

de Bary: The micro-organisms of the bowel *A. f. exp. Path.* xx **1886** (with references)
Bollinger: *Zur Aetiol. d. Infectionskrankheiten* Munich **1881**
Bouisson: A case of intestinal anthrax *A. de méd. exp.* I **1889**
Fischl: Haemorrhagic enteromycosis *A. f. exp. Path.* xvi **1883**
Friedberger and Fröhner: *Lehrb. d. spec. Path. u. Therap. d. Hausthiere* Stuttgart **1885** (translated by Zuill) Philadelphia **1895**
Gärtner: The meat-poisoning epidemic at Frankenhausen *Breslau. ärztl. Zeit.* **1888**
Huber: The Wurzen epidemic *A. d. Heilk.* xix **1878**
Leube and Müller: *D. A. f. klin. Med.* xii **1874**
Nauwerck: Sausage-poisoning *Münch. med. Woch.* **1886**
Quincke: Meat-poisoning *Mittheil. d. Vereins Schleswig-Holsteinischer Aerzte* 10 **1885**
Raimbert: *Traité des maladies charbonneuses* Paris **1859**
Schmidt-Mühlheim: *Handb. d. Fleischkunde* Leipzig **1884**
Straus: *Le charbon des animaux et de l'homme* Paris **1886**, and *A. de physiol.* I **1883**
Vierhuff: *Ueber Anthrax intestinalis* Dorpat **1885**
Walder: Meat-poisoning *Berl. klin. Woch.* **1878**
Waldeyer: Intestinal mycosis *V. A.* 52 **1871**
Zangger: *A. f. Thierheilk.* xxiv Zürich **1871**

208. **Tumours of the intestine.** As we have already stated when discussing the inflammatory affections of the intestine, the mucosa and submucosa are not infrequently the seat of inflammatory and hyperplastic growths involving both the connective

tissue and the epithelium. These growths are usually secondary to necrotic and inflammatory processes, some of them of an infective nature, though they sometimes arise without any demonstrable cause.

Proliferous connective tissue usually reproduces tissue of the same kind, and gives rise either to circumscribed fibrous excrescences, or more frequently to thickening and induration of the mucosa and submucosa. In certain cases the chief product is lymphadenoid tissue, generally taking the form of hyperplasia of the lymph-follicles (Fig. 338), but sometimes intercalated in the submucous connective tissue outside the follicles. Such hypertrophic swellings are at times produced in the course of infective diseases, such as diphtheria, but they are often of merely local origin. They reach their greatest luxuriance in the affections described as simple and leukaemic **adenia** (Art. 38), whose causation is still unknown. In these affections the follicles are replaced by bulky nodes and swellings, and by broad flattened lymphadenoid patches, which on section have a marrowy pink or greyish-white appearance.

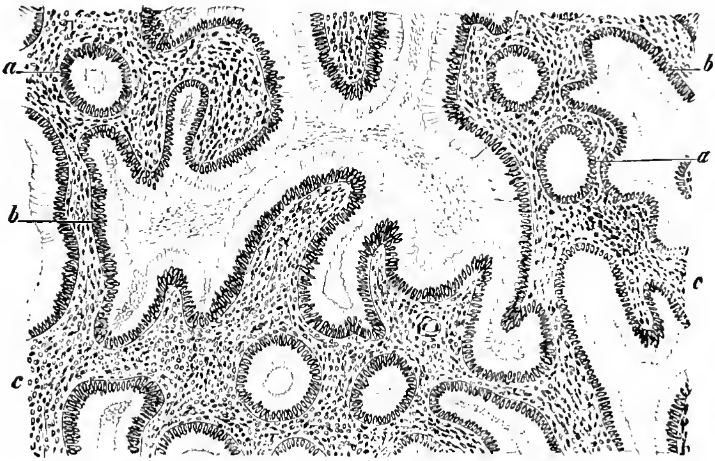

FIG. 350. GLANDULAR POLYPUS OF THE INTESTINE.

(*Preparation hardened in alcohol, stained with alum-carmine, and mounted in Canada balsam* : × 80)

a transverse section of glandular tubules
b longitudinal section of branching tubules
c stroma with abundant cells

Proliferous epithelium reproduces both the ordinary surface epithelial cells and new glands (Art. 200, Fig. 340), the multiplication of the cells taking place chiefly in the deeper layers of the crypts of Lieberkühn. The process is often obviously of a regenerative kind, as when the borders and floors of ulcers (Fig. 340) become covered over with gland-containing tissue. Not infrequently however the formation of new tissue is so abundant as to lead to hyperplastic overgrowth, circumscribed thickenings of the mucous membrane and polypous excrescences being produced (Fig. 342 *c*), which from the numerous glands they contain are usually described as **glandular polypi.** They are commonest in parts of the large intestine that have been the seat of antecedent dysenteric processes, but they are also met with in the small intestine, and sometimes appear in parts that show no signs of previous inflammation.

The glands of such polypi are simple or branching tubules, lined with tall columnar epithelium (Fig. 350 *a b*), and some of them may be dilated into **cysts** by the retention of secretion. The glandular tubules are not entirely typical in structure, though in the benign growths they are generally provided with only a single layer of cylindrical epithelial cells.

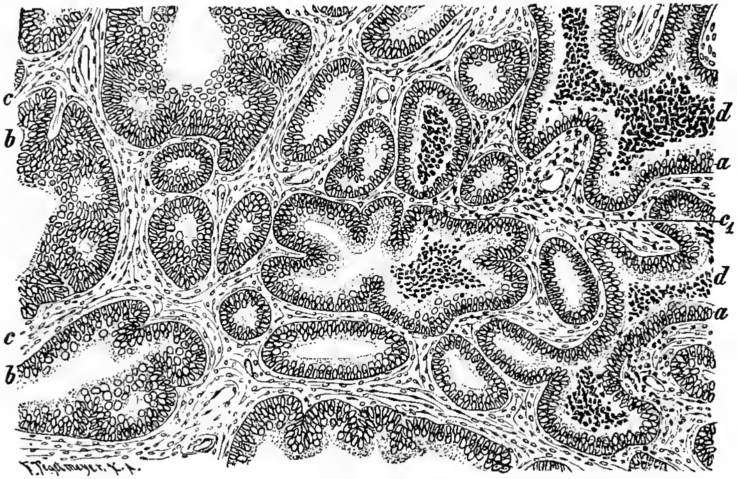

FIG. 351. ADENO-CARCINOMA OF THE RECTUM.

(*Section prepared as in* FIG. 350 : × 80)

a branching glandular tubules lined with many-layered epithelium
b tubules with proliferous epithelium and papillary outgrowths
c c_1 stroma
d aggregations of round-cells

209. Every variety of **carcinoma** to which mucous membranes are subject appears in the intestinal tract. The various forms are described as solid medullary cancer, adenomatous cancer, colloid cancer, and scirrhus, the adenomatous form (adeno-carcinoma or malignant adenoma) being the commonest. The diagnosis of the latter from benign glandular polypus rests partly on the more luxuriant and atypical overgrowth of its epithelium (Fig. 351 *a b*), and partly on its tendency to infiltrate the contiguous tissue. It should however be remarked that these two characters are not necessarily coincident in every case: growths

whose tubules are all lined with a single-layered cylindrical epithelium, and which therefore are still non-malignant, sometimes penetrate the *muscularis mucosae* and invade the submucosa, thus in some degree simulating the appearance of a malignant or carcinomatous growth.

The rectum (Fig. 353), the sigmoid, splenic, and hepatic flexures (Fig. 352) of the colon, and the caecum, are the commonest seats. Rectal cancer sometimes extends only to the parts about the anus, but in other cases it infiltrates the intestine for a length of ten centimetres or more (Fig. 353 *a*).

Carcinoma of the small intestine is rare, but in the duodenum, and especially in the neighbourhood of the opening of the bile-duct, it is somewhat more frequent.

Intestinal cancer takes the form of soft fungous tumours which are solitary and sharply circumscribed or spread over a considerable area (Fig. 352 *a*). Infiltration of the intestinal wall with cancer-cells usually takes place at an early stage, and leads to thickening and induration (Fig. 353 *a*). If this extends round the whole circumference of the bowel, it is transformed into a thick-walled rigid tube; the rectum is the commonest seat of this indurative change, and less frequently the colon.

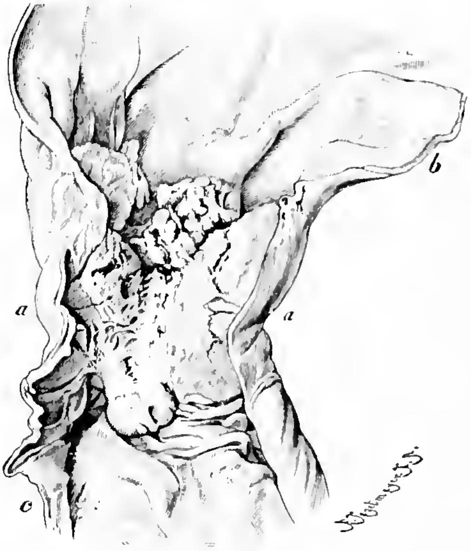

FIG. 352. ANNULAR DIFFUSE FUNGOUS CANCER OF THE HEPATIC FLEXURE OF THE COLON.

(*Seven-tenths of the natural size*)

a cancerous portion *b* part above, and *c* part below, the cancer

On post-mortem examination of most cases of this disease we find the surface of the neoplasm already broken down, leaving a cancerous ulcer with characteristically infiltrated borders. But sometimes the borders likewise are disintegrated and eroded, and then the ulcer has quite the appearance of an ordinary non-malignant inflammatory ulcer. In other cases the borders and floor of the ulcer become scarred over and shrunken, leading sometimes to extreme constriction of the bowel: this is particularly apt to occur when the ulceration extends in an annular form round the intestine (Fig. 353 *a*).

When a cancer of the intestine breaks down and ulcerates, at the same time invading the deeper layers of the wall, it generally induces inflammatory changes in the serous coat. These lead to the formation of new vascular fibrous tissue, by which the affected part of the bowel is bound down to the surrounding structures. Perforation of the intestine occurs in some cases as a result of cancerous ulceration. Metastatic growths are met with chiefly in the lymph-glands, peritoneum, and liver.

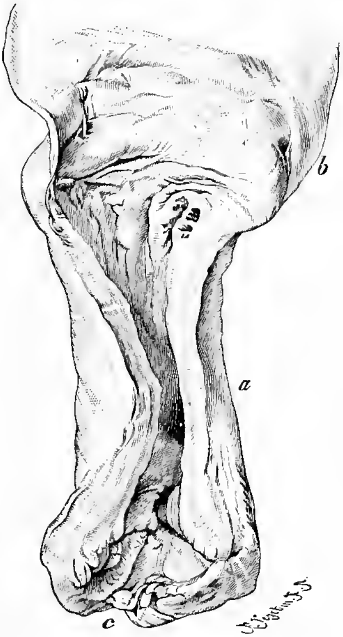

FIG. 353. CANCER OF THE RECTUM.

(*One-half the natural size*)

a thickened, indurated, and strictured portion of the rectum
b dilated portion of the rectum
c anus

Connective-tissue growths are rare in the intestine, and are of much less importance than the carcinomata. Fibroma and lipoma, more rarely myoma, angioma, and sarcoma, have been observed. These are developed from the mucosa and submucosa, and in part from the muscular and serous coats. When they protrude as polypi from the inner surface of the bowel they may obstruct the passage, or by their weight drag down and invaginate a portion of the wall. Pedunculated growths may thus be actually torn off by the peristaltic movement of the bowel, and so be extruded with the faeces.

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BARDENHEUER: Multiple glandular growths of the mucous membrane of the large intestine with carcinoma of the rectum *A. f. klin. Chir.* XLI **1891**
BÖTTCHER: Morbid anatomy of the lungs and intestine in leukaemia *V. A.* 37 **1866**; Myoma *V. A.* 104 **1886**
DU CASTEL: Cancer of the ileum *A. gén. de méd.* **1882**
ESMARCH: Diseases of the rectum and anus *Deutsche Chirurgie* part 48
FRIEDREICH: Leukaemia *V. A.* 12 **1857**
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HOLTKAMP: *Die Polypen d. Mastdarmes* **1886**
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LANGHANS: Glandular polypus of the ileum *V. A.* 38 **1867**
LAVERAN: Diagnosis of polypus from micro-carcinoma *A. de physiol.* III **1876**
LODE: Myoma of the ileum *Wien. klin. Woch.* **1894**

LUBARSCH: Primary cancer of the ileum *V. A.* 111 **1888**
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PICK: Primary sarcoma of the small intestine *Prag. med. Woch.* **1884**
PRZEWOSKI: Chylangioma of the mucosa and submucosa *Cent. f. allg. Path.* I **1890**
ROSENHEIM: *Art.* Intestinal tumours *Eulenburg's Realencyklop.* **1894** (with references)
SHEILD: Large polypus of the rectum *Trans. Path. Soc.* XXXIX London **1887**
STERN: Primary sarcoma of the small intestine in infants *Berl. klin. Woch.* **1894**
TAYLOR: Lymphoma of the small intestine *Trans. Path. Soc.* XXVIII London **1887**
VIRCHOW: *Die krankhaften Geschwülste* II Berlin **1865**
WALDEYER: Development of carcinoma *V. A.* 41 **1867**, and 55 **1872**
WHITEHEAD: Large multiple polypus *B. M. J.* I **1884**

210. Of **animal parasites** found in the intestine the following may be enumerated. They are fully described in the volume on General Pathological Anatomy.

(1) *Cercomonas intestinalis*, *Paramoecium coli*, *Amoeba coli mitis*, and *Amoeba dysenteriae*
(2) *Taenia mediocanellata* or *saginata*
(3) *Taenia solium*
(4) *Taenia nana*, *Taenia cucumerina*
(5) *Bothriocephalus latus*
(6) *Ascaris lumbricoides*, *Ascaris mystax*
(7) *Trichina spiralis*
(8) *Trichocephalus dispar*
(9) *Oxyuris vermicularis*
(10) *Anchylostoma duodenale* or *Dochmius duodenalis*
(11) *Anguillula stercoralis* and *A. intestinalis*

Of **foreign bodies** met with in the intestine, the only ones which have any pathological interest are those that are produced *in situ*.

To say nothing of the hard solid scybalous masses produced by the retention of faeces, there are certain stony concretions which have received the name of **enteroliths** (Fig. 354) or intestinal calculi. They are found chiefly in the caecum, vermiform appendage, and colon, and more rarely in the small intestine; they usually lie in sacculations or diverticula of the wall. Three varieties have been distinguished.

(1) Heavy stony stratified concretions, the successive layers being white, yellow, and brown. These consist of magnesium phosphate, ammonio-magnesium phosphate, and organic matters. They are seldom larger than a Spanish chestnut, and are generally rounded in shape. They frequently contain some small foreign body as a nucleus.

(2) Enteroliths of low specific gravity and irregular form, porous and somewhat elastic in texture. They consist of a felted mass of indigestible husks and other vegetable refuse, intermingled with indurated faeces and earthy or chalky matters.

(3) Stones due to the long-continued use of certain mineral drugs, such as chalk, magnesia, and bismuth oxide.

In addition to these, which are formed in the intestine itself, we sometimes find **gall-stones** which have escaped from the bile-ducts.

Such concretions, and foreign bodies reaching the intestine from the exterior, may give rise to partial or complete obstruction of the bowel. This frequently follows from the lodgment of bodies in one of the pouches of the rectum. The result of course is stoppage of the faeces, and with this sometimes inflammation, ulceration, and perforation of the intestinal wall.

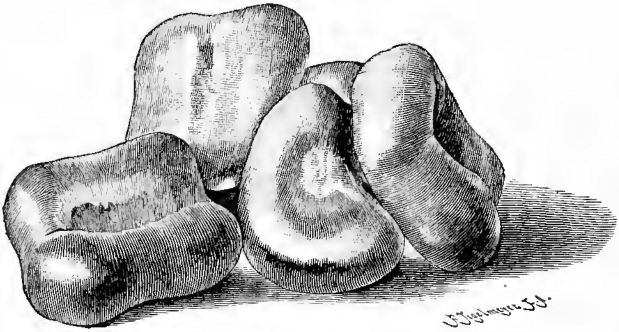

FIG. 354. FACETTED INTESTINAL CONCRETIONS.
(*From a case of perityphlitic abscess: natural size*)

In horses and horned cattle intestinal calculi are met with far more frequently than in human beings. The intestines of these animals always contain fragments of undigested vegetable refuse and hairs which have been licked off and swallowed, and such matters form a nucleus for concretions to grow about. The true calculi, met with chiefly in horses, are hard stony balls consisting for the most part of magnesium phosphate. The spongy or false stones consist of felted hairs and fibres that are merely crusted over with salts. Balls are sometimes found composed entirely or almost entirely of hairs; these go under the name of hair-balls or **bezoars**, or aegagropili (from αιξ αγριος a wild goat, πιλος felt). In ruminants they usually lie in the caecum or colon, in swine they are more frequently found in the small intestine.

SCHUBERG asserts that the intestinal concretions of the herbivora consist chiefly of carbonates, while those of the carnivora are phosphatic. In man the composition of these stones varies with the food habitually used.

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CHAPTER LXV

THE PERITONEUM

211. The **peritoneum** is a fibrous membrane, covered with a single layer of flat cells, which lines the abdominal cavity and invests the viscera contained within it.

The affections of the peritoneum are for the most part consecutive to diseases of the abdominal organs; but certain morbid processes are chiefly or entirely confined to the membrane itself, and in some cases originate in it.

Peritoneal **haemorrhages** occur in the form of petechiae, ecchymoses, large suggillations, haematomata, and haemorrhagic infarcts. They may be caused by traumatic injury, inflammation, changes in the walls of the blood-vessels, vascular obstruction or occlusion, the haemorrhagic diathesis, and venous engorgement. In the subserous cellular tissues, especially about the kidneys and pancreas, spontaneous rupture of the blood-vessels sometimes takes place, leading to such copious haemorrhage that death ensues.

The effusion of blood into the abdominal cavity is usually consequent upon injury and laceration of the viscera, spontaneous haemorrhage from the ovaries and tubes, or extrauterine pregnancy: it is often so abundant as to endanger life.

The new blood-vessels developed in the course of peritoneal inflammation, of tuberculosis, and of the growth of tumours, are very apt to give way and bleed, the extravasation taking place into the tissues or into the abdominal cavity.

Extravasations into the tissues have when recent a red tint, but after a time they become brown or slate-coloured. Blood which has escaped into the abdominal cavity is largely re-absorbed, the liquid portions directly and speedily, the coagulated matters more slowly. These last are gradually broken down and liquefied before they are taken up by the lymphatics, and they usually set up around them some inflammatory proliferation of the peritoneum, the effect of which is to facilitate the process of resorption.

Ascites is the name given to a collection in the abdominal cavity of liquid which is not due to antecedent inflammation. It is generally a concomitant of diseases of the heart, liver, and kidneys, and emphysema of the lungs, the cause of the exudation being venous engorgement and the resulting changes in the vessels. It is also a frequent accompaniment of abdominal tumours.

According to Quincke, ascites occasionally makes its appearance in girls about the time of puberty, without any apparent cause, and disappears as soon as menstruation is established.

The effused liquid in ascites is poor in formed elements, containing only a few cast-off endothelial cells, which generally enclose fat-globules or exhibit other signs of disintegration, together with a small number of swollen lymphoid cells. The liquid is clear, colourless, or slightly yellow, and may or may not contain delicate gelatinous flakes of fibrin. A more extensive admixture of fatty cells may give it a slight milky turbidity (chylous dropsy, *hydrops lacteus*). When jaundice is present the liquid may be stained with bile; effusion of blood gives the liquid a reddish tinge.

When some direct communication exists between the abdominal lymph-vessels and the abdominal cavity, the transuded liquid sometimes assumes a white milky appearance, resembling that of chyle, and the condition is spoken of as **chylous ascites**. It is liable to supervene in cases of laceration of the thoracic duct, in extreme lymphatic engorgement from obstruction, in filarial disease, and in abdominal lymphangiectasis.

The immediate effect of ascites is distension of the abdomen and increase of the intra-abdominal pressure. Extreme ascites presses up the diaphragm and impedes the breathing. In certain cases the umbilical region is forced out and sometimes even becomes gangrenous, so that actual rupture takes place.

When the effusion persists for a long time, turbid whitish patches, thickenings, and adhesions between adjacent parts, are produced in the peritoneum. The turbid patches are due chiefly to endothelial catarrh, whereby the endothelial cells swell, subdivide, and are cast off, some of them undergoing fatty changes. The underlying connective tissue is often infiltrated with leucocytes, and its fixed cells become proliferous. The result is fibrous hyperplasia, with the formation of the thickenings and adhesions referred to.

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212. Acute inflammation of the peritoneum, or **acute peritonitis**, usually starts from one of the abdominal organs, and is thus in the first instance a local disease; but it extends very readily over the entire peritoneum, especially when intense irritants or pathogenic bacteria capable of multiplication gain access to the abdominal cavity. In rare instances the affection is traceable to haematogenous infection. The severer forms of purulent, seropurulent, or sero-fibrinous inflammation are most commonly produced by pyogenic *Streptococci*, though *Staphylococci*, *Diplococcus pneumoniae*, and *Bacillus coli* are capable of giving rise to these inflammations. The supervention of infection is favoured by the presence in the abdominal cavity of blood, fibrin, transudations, faeces, and dead or dying tissue.

The common causes of peritonitis are — various traumatic injuries; puerperal infection of the genital system; perforation of the stomach or intestine, or of the vermiform appendage, with escape of faeces; inflammations of the intestine, stomach, liver, gall-bladder, internal genital organs, bladder, pancreas, kidneys, spinal column and pelvis, pleura, and diaphragm; ulcers of the intestinal canal which extend to the serosa; and lastly, gangrene of the intestine, omentum, or of any other organ lying within the abdominal cavity, as in cases of strangulated hernia, intussusception, volvulus, or occlusion of the abdominal arteries.

Peritonitis in new-born infants not infrequently starts from an inflamed or gangrenous navel.

Acute diffuse haematogenous peritonitis occurs most commonly in connexion with nephritis, pyaemia, rheumatic polyarthritis, and the acute exanthemata: peritonitis from this class of causes is however much less frequent than that originating in the abdominal organs and the parts adjacent. It is extremely rare for peritonitis to be the only symptom of an infection.

Sero-fibrinous and haemorrhagic peritonitis in new-born infants suffering from inherited syphilis is by no means rare; but this form of inflammation cannot be regarded as pathognomonic of syphilis, for the same condition may arise and prove fatal in infants that are not syphilitic.

The character of the inflammation is largely determined by the exciting cause. Peritonitis following perforation of the intestine and stomach, intestinal gangrene, rupture of hepatic and splenic abscesses, and purulent parametritis, usually assumes a purulent character; and if no adhesions have been previously formed in the abdomen, it generally involves the entire peritoneum. The hyperaemic serosa, studded here and there with small haemorrhages, becomes covered over with yellowish-white liquid or creamy pus; when the exudation is more abundant the pus collects in the dependent parts of the abdominal cavity, which may at length become distended with purulent liquid.

The coils of intestine are more or less displaced and compressed, while their walls are sodden and easily torn. When the bowel is handled the serosa readily separates from the muscular coat. Very frequently the several coils are glued together, the free surfaces of the peritoneum being covered with delicate yellowish-white flakes (described by surgeons as 'coagulable lymph'), which are simply fibrinous coagula interspersed with pus-corpuscles. When the peritonitis is due to perforation of the intestine, the pus is discoloured and often mingled with faecal matter, the odour is extremely offensive, and the upper portion of the abdomen frequently contains gas, which escapes with a hissing noise when the cavity is opened.

Milder forms of peritonitis are associated with inflammations and with non-perforating abscesses and ulcers of the intestine, liver, spleen, internal genitals, perinephric tissue, and bladder; and according to the intensity of the irritant lead to circumscribed or diffuse exudations which are sero-purulent, sero-fibrinous, or purely fibrinous in character, and occasionally to small haemorrhages. At the outset the peritoneal tissue is hyperaemic (Fig. 355 *c d*) and infiltrated with liquid and immigrant leucocytes (*e*), while the endothelium swells and undergoes proliferation (f_1) or desquamates and breaks down (*f*). In fibrinous inflammations it is apt to be converted into homogeneous denucleated flakes. Later on the surface is covered over with exudation.

Severe general peritonitis usually ends in death. Local collections of pus may rupture externally through the umbilicus or into contiguous coils of intestine, into the stomach, pleura, or pericardium, or into the bladder and vagina.

Recovery takes place, after resorption or evacuation of the exudation, by the production of a thin, translucent, and highly-vascular granulation-tissue which is subsequently converted into firm connective tissue. In this manner fibrous deposits are formed on the serous surface, and adhesions between contiguous organs are set up (*peritonitis adhaesiva*). The connexions consist of fine bands and membranes, or of dense unyielding fibrous tissue, which effectually prevents any relative movement of the adherent parts. In this way the stomach may become more or less firmly attached to the under surface of the liver; the upper surface of the latter organ to the diaphragm; the spleen to the fundus of the stomach, the diaphragm, and the external abdominal wall; the omentum to the abdominal wall and the adjacent viscera; and the intestinal coils to each other: while bands and membranes pass from the uterus and ovaries to various portions of the parietal peritoneum or to adjacent parts of the bowel. Contraction and induration of the omentum frequently take place in the region affected by the inflammation, and its several surfaces become firmly bound together. The mesentery of the intestine when thus thickened is often shrunken and shortened. In rare instances the peritoneal investment of the stomach or intestine undergoes such induration and contraction that the affected parts of the tract cease to be extensible (*peritonitis deformans*).

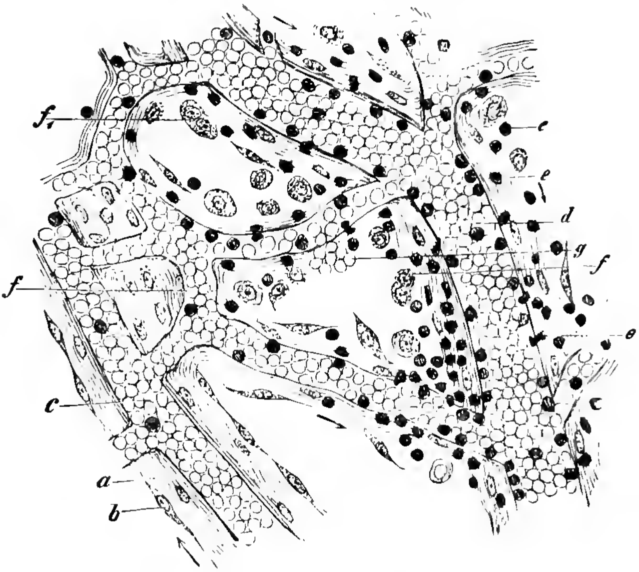

FIG. 355. INFLAMED OMENTUM FROM THE HUMAN SUBJECT.

(*Preparation fixed with perosmic acid and mounted in glycerine*: × 200)

a	normal fibrous trabecula
b	normal epithelium
c	small artery
d	vein with colourless blood-corpuscles peripherally disposed
e	colourless blood-corpuscles migrated or migrating
f	desquamated epithelium commencing to break down
f_1	proliferous epithelium
g	migrated red blood-corpuscles

Chronic peritonitis is generally a later stage of acute inflammation, and like the latter may be local or involve the whole peritoneum. Residual exudations from an antecedent acute inflammation, or special sources of irritation, such as an abscess due to perforation of the vermiform appendage, are liable to keep up or to renew the peritoneal inflammation, and in this way the production of indurations and adhesions is maintained or increased. In like manner infective peritonitis is apt to be rekindled from time to time, the original exciting agent surviving in a quiescent state, but still capable of reproduction and multiplication. This recurrent activity appears to be characteristic of gonorrhoeal perimetritis and parametritis, but it is not unknown in connexion with affections due to other micrococci, such as *Streptococcus pyogenes*.

In rare cases after the subsidence of diffuse peritonitis exudation and fibrous proliferation now and again recur in the peritoneal adhesions and thickenings, leading to fresh indurations, adhesions, and contractions (*peritonitis deformans*). In this manner the whole of the intestinal coils may become knotted into a shrunken mass that occupies only a small portion of the abdominal cavity, the remainder of the space being filled with liquid. The peritoneum in such cases is generally converted into a thick white lustrous membrane.

Lastly, chronic or slight but recurrent acute inflammation is often associated with chronic venous engorgement of the abdominal viscera (Art. 211), such as is often due to cirrhosis of the liver, or to the growth of large tumours. This form like the others is capable of causing indurations and adhesions of the serous surfaces. It is very rare indeed for chronic non-tuberculous peritonitis to begin insidiously, without a preliminary acute stage, or without a discoverable exciting cause in some visceral disease or vascular engorgement.

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213. **Tuberculosis** of the peritoneum is either of haematogenous origin, and is then usually associated with more or less extensive miliary tuberculosis, or a lymphogenous affection transmitted from a tuberculous focus in some abdominal viscus or in the adjoining tissues. In rare cases the affection of the peritoneum appears to be primary, the mode of entrance of the virus being undiscoverable.

When the tubercle-bacilli are conveyed to the abdominal vessels by the blood, an eruption of grey tubercles takes place in the peritoneum, but no perceptible change in its tissue immediately ensues, even the endothelial stratum overlying the tubercles remaining intact.

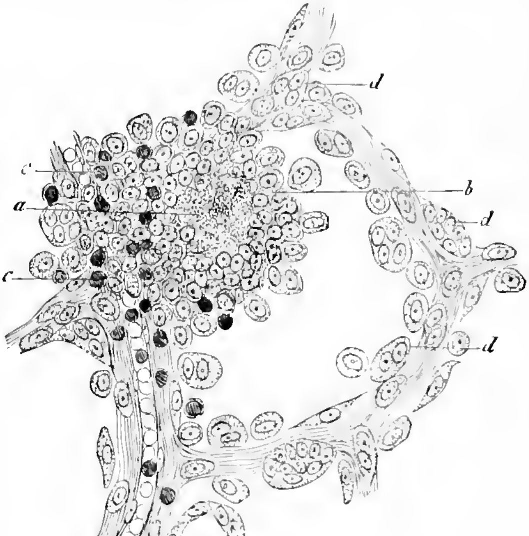

FIG. 356. TUBERCULOSIS OF THE OMENTUM.

(*Carmine-staining*: × 200)

a caseous centre of the tubercle
b epithelioid cells
c lymphoid cells
d proliferous epithelial cells in the neighbouring tissue

When the bacilli reach the peritoneal lymphatics from the intestine or other viscus, a very limited eruption of tubercles and tuberculous nodes appears, and generally follows the course of the affected vessels. The tubercles are surrounded by a hyperaemic areola, within which at a later stage vascular granulomatous tissue is developed.

If the bacilli gain access to the abdominal cavity itself, the eruption of tubercles spreads rapidly over the greater part or the whole of the peritoneum; this soon becomes more or less thickly studded with tuberculous nodules, and severe general peritonitis usually ensues. The membrane is hyperaemic, the endothelium becomes proliferous (Fig. 356 *d*) and desquamates freely, and from the vessels leucocytes are extravasated and a more or less copious liquid exudation is poured out. The latter often contains red blood-corpuscles, which give the effusion a blood-stained appearance, and cause the serous surface to be flecked with reddish ecchymoses, that afterwards become slaty-grey in colour.

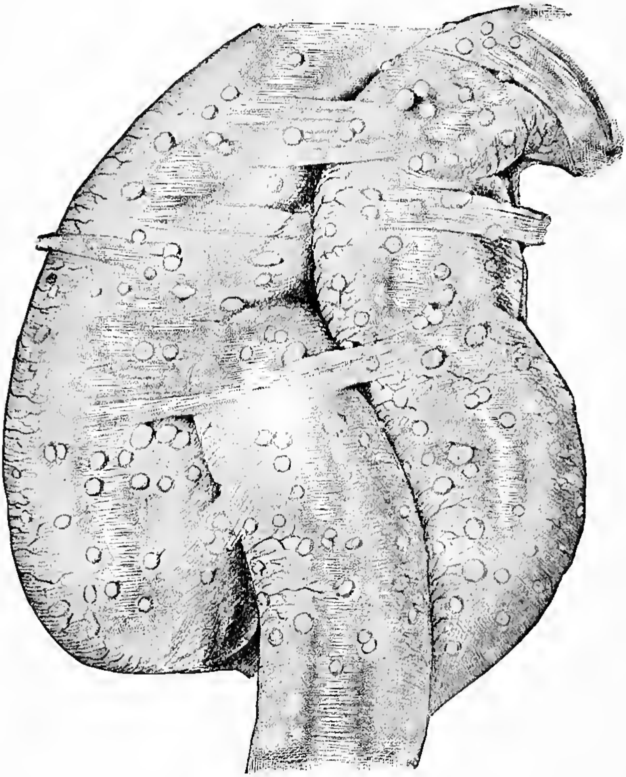

FIG. 357. ADHERENT COILS OF INTESTINE STUDDED WITH TUBERCLES.
(*Seven-eighths of the natural size*)

Solitary foci of infection sometimes subside and recover; but as a rule, unless death intervenes, the morbid process continues to advance. Larger tubercles and nodes develope (Fig. 357), and presently broad caseous patches are produced on the serous surface of the viscera. Fibrous adhesions (Fig. 357) are formed between apposed peritoneal surfaces, and the omentum is beset with single or aggregated tubercles, becoming by degrees thickened and contracted, till it looks like a stiff unyielding flap overlying the bowels, or a thick rope drawn across them. The mesentery becomes indurated and shrunken, and it also is more or less thickly beset with tubercles.

The liquid effusion varies much in quantity, and in appearance is serous, sero-purulent, haemorrhagic, purulent, fibrino-purulent, or even purulent and putrid. In the latter case the infection is probably always of a mixed kind; but it is still a matter of dispute whether the multiple infection existed *ab initio*, or whether the microbes of suppuration and of putrefaction gain access to the abdominal cavity from the bowel subsequently to the tuberculous infection. When the process continues for any considerable time without suppuration, the agglutination of the intestinal coils is often so complete that they are matted together into a single coherent mass.

In rare instances the intestine becomes perforated at one or more points, particularly when tuberculous ulceration of the mucous surface coexists with the peritoneal affection.

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214. **Primary neoplasms** are not often met with in the peritoneum. The most interesting are the tumours hitherto described as **endotheliomata**, which generally take the form of multiple flattened nodular growths, white in colour, and either coalescent or connected by neoplastic bands; more rarely they take the form of large isolated tumours of marrow-like consistence, the peritoneum about them being more or less thickened. They are usually accompanied by a certain amount of serous or sero-fibrinous blood-stained exudation.

The tumour is characterised by the presence in it of nests and clusters of endothelial cells, which lie in a dense fibrous stroma and follow exactly the course of the lymphatic vessels. They arise from the multiplication of the endothelial cells of the serous surface or of the lymphatics.

Lipoma, fibroma, myxoma, and sarcoma are rare in the peritoneum. Lipomata arise most frequently as tumour-like enlargements of the *appendices epiploïcae* of the colon. Pedunculated lipomata may be separated from their attachments and form free peritoneal bodies which subsequently undergo calcification. WALDEYER has described a plexiform angiosarcoma of the abdominal cavity, and WEICHSELBAUM a lymphangioma of the mesentery.

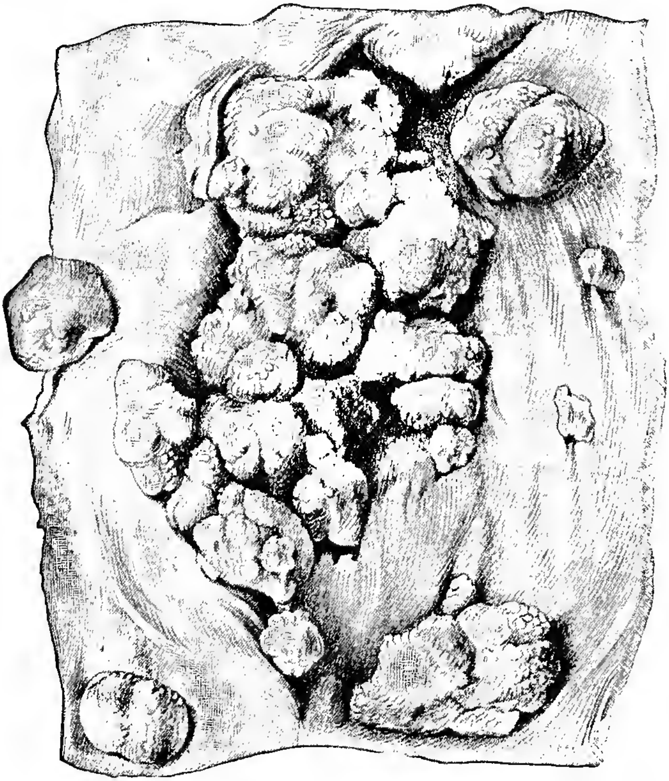

FIG. 358. PAPILLOMATOUS CANCER OF THE UNDER SURFACE OF THE DIAPHRAGM.

(*Due to metastasis from a cancerous papilliferous cystoma of the ovary*)

Tumours of the subserous tissue are more common, especially fibroma, lipoma, and sarcoma.

Of **secondary growths** carcinoma is the most frequent. It generally originates in one of the abdominal viscera, and as a rule takes the form of isolated nodes and nodules. Cases however occur in which within the space of a few weeks the entire peritoneum is beset with miliary growths resembling tubercles, or with large marrowy (Fig. 358) or firm excrescences. The tissue about the growths may be but little altered, or it may be hyperplastic, indurated, and highly vascular. When the nodes are closely aggregated, the affected portion of the serous membrane (such as the omentum) is liable to be converted into a thick tuberous mass.

In a few cases the carcinomatous infiltration is more indefinite or diffuse, especially in connexion with colloid cancer of the intestine or the ovary. When this form invades the peritoneum it sometimes causes the entire abdominal cavity to be filled with gelatinous masses.

Scirrhous cancers also are apt to induce diffuse cicatricial thickening of the peritoneum and subperitoneal tissue, without any formation of metastatic nodes. Such nodes are at times produced by sarcoma, but not very frequently.

Peritoneal **dermoids** and complex **teratomata** (foetal inclusions) develope in women from the ovary or its neighbourhood, though they appear at times in other situations.

Serous cysts are generally met with in connexion with the female generative organs (Art. 317). Occasionally peritoneal cysts of various sizes occur in other parts of the abdomen, some of which develope in membranous adhesions, while others arise independently of antecedent inflammation. Cases are recorded in which the abdominal cavity was more or less filled with pedunculated thin-walled vesicles, varying in size from that of a pea to that of a walnut, and grouped in clusters like bunches of grapes. Some of these growths are lymph-cysts due to dilatation of the lymphatics; others are of the nature of cavernous lymphangiomata.

Of the **animal parasites** affecting the peritoneum the *Echinococcus* is of some importance. It may form hydatid cysts of considerable size, and the cysts are often bound to the surrounding parts by fibrous adhesions. *Cysticerci* are occasionally met with, but they seldom cause any notable disturbance. Sometimes intestinal parasites, such as round-worms, escape into the peritoneal cavity either through some pre-existing wound of the intestine or by active penetration of its wall.

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SECTION X

THE LIVER AND PANCREAS

CHAPTER LXVI

MALFORMATIONS AND MALPOSITIONS OF THE LIVER

215. **Malformations** of the liver are not common, and have little clinical importance. Absence of the organ is very rare, at least in foetuses which otherwise present no important anomaly. More frequent are congenital deviations from the normal shape, such as absence of particular lobes, or abnormal lobulation. In a few cases **accessory livers** have been noted, in the form of small nodules situated in the suspensory ligament. Absence of the gall-bladder has more than once been observed, as also congenital stenosis or dilatation of the biliary passages, and anomalies in the opening of the common bile-duct into the intestine.

Among congenital anomalies of position the most notable are transposition of the liver to the left side, in *Situs viscerum transversus*, and protrusion of the organ into the thorax in cases of saccular bulging or defect of the diaphragm, or to the exterior when the abdominal wall is cleft.

Acquired **deformities** are very frequently the result of morbid textural changes in the hepatic parenchyma, or in the neighbouring organs. Thus tight lacing, by which the lower part of the thorax is violently compressed, gives rise to a characteristic deformity of the liver. In this condition the part of the organ adjacent to the costal margin is indented and depressed, its fibrous capsule is white and thickened, and the underlying liver-tissue is atrophic, many of the lobules having completely disappeared. When the indentation is deep, the right lobe appears as if divided into a larger upper lobe and a smaller lower one; in extreme cases the lower part becomes freely moveable on the upper, sometimes turning upward in front of it as on a hinge.

The outer surface of the right lobe is frequently marked with shallow grooves corresponding to the course of the ribs. Oftener still the upper surface shows antero-posterior grooves or furrows produced by the pressure of projecting hypertrophied muscular bundles of the diaphragm where it overlies the liver. These have been termed **diaphragmatic grooves.** ORTH has however seen similar furrows in a seven-months foetus, and thinks that in some cases at least they have a different origin.

Acquired **displacements** of the liver are not uncommon, and are generally due to rotation of the organ on its transverse axis, so that the level of the anterior margin varies considerably with the varying degree of distension of the abdomen. Much rarer is abnormal depression of the whole organ, with extension of the suspensory ligament (*hepar mobile* or **floating liver**). This condition is most marked in cases of extreme dilatation of the stomach, accompanied by great relaxation of the anterior wall (ventral hernia). The liver is also apt to be forced downwards when the right side of the diaphragm is depressed by pleuritic effusion or pneumothorax.

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HASSE: The movements of the diaphragm and their effect on the abdominal organs *A. f. Anat. u. Phys.* **1886**, and *Anat. Anzeiger* I **1886**

KRANOLD: Cases of floating liver *Inaug. Diss.* Tübingen **1884**

LANDAU: *Die Wanderleber und der Hängebauch der Frauen* Berlin **1885**

LEGG: Moveable liver *St Barth. Hosp. Reports* XIII London **1877**

LEOPOLD: Hepar mobile *A. f. Gynäk.* VII **1874**

LIEBERMEISTER: *Beiträge z. path. Anat. u. Klinik der Leberkrankheiten* Tübingen **1864**

MÜLLER: Floating liver *Berl. klin. Woch.* XIX **1882**

SABOURIN: *Rech. sur l'anat. norm. et pathol. de la glande biliaire* Paris **1888**

SEAGER: Displacement of the liver *B. M. J.* II **1885**

THIERFELDER: Hepar mobile *Ziemssen's Cyclopaedia* IX New York **1874–80**

THOMSON: Anatomical variations *Journ. of Anat.* XIX London **1884**

VIOLA: Malformations of the gall-bladder *Lavori dell' Ist. anat. patol. di Perugia* **1890**

WINKLER: Hepar mobile *A. f. Gynäk.* IV **1872**

CHAPTER LXVII

SIMPLE HEPATIC ATROPHY

216. **Atrophy** of the hepatic tissue may be due either to general disorders of nutrition or to local disease of the liver. In many cases atrophy is a secondary result of degenerative processes affecting the parenchyma (Arts. 221–224), induced by disturbance of the circulation, poisons, infections, and the like. But there is also a form of simple atrophy in which the hepatic tissue is gradually reduced in bulk, without undergoing any striking change in its structure.

The average weight of the liver in the adult is about 1500 to 1600 grammes. In the condition of senile marasmus, the weight may be reduced to less than half this amount. In these cases the organ is as a rule uniformly diminished; but the atrophy is generally most marked on the anterior margin, and is not infrequently more pronounced in one lobe than in the other. When the wasting is considerable the gall-bladder projects beyond the anterior edge of the liver, which thus appears to be retracted, and the margin seems often reduced to little more than a membrane. The surface of the liver is in some instances smooth, in others slightly granular; the parenchyma is generally of a pale-brown colour.

Senile atrophy consists essentially in a progressive dwindling of the liver-cells (Fig. 360 *A*); in the marginal portions those lying just beneath the serous covering sometimes disappear so completely that nothing remains of entire lobules save their connective-tissue framework and vessels. As a rule the framework collapses as the atrophy proceeds, and is reduced to a kind of loose fibrous tissue containing few blood-vessels (Fig. 359 *e*). In the end the parenchyma consists of nothing but this fibrous tissue with the capsule of Glisson (*d*) and the blood-vessels and bile-ducts it encloses. While the atrophy is proceeding the liver-cells are often beset with yellow or brownish pigment (Fig. 360 *A*), consisting of bile-pigment or of haemosiderin.

When the supply of nutriment to the organism is for any reason interfered with, so that general wasting takes place by starvation, atrophy of the liver with progressive diminution in the size of its cells is induced. The experiments of BIDDER, Schmidt, and Voit have shown that in the case of dogs and cats starvation may reduce the bulk of the liver by two-thirds.

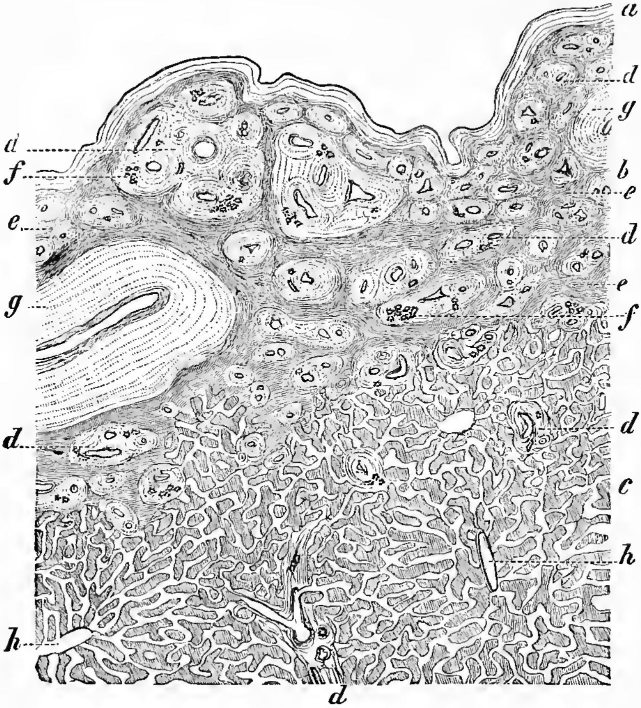

Fig. 359. Section from the margin of a much atrophied liver.

(*Preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam:* × 30)

a peritoneal serous membrane
b completely atrophied liver-tissue
c normal lobules
d portal sheaths (capsule of Glisson) enclosing blood-vessels and bile-ducts
e loose fibrous tissue taking the place of lost hepatic parenchyma
f bile-ducts
g larger portal veins
h intralobular (central) veins

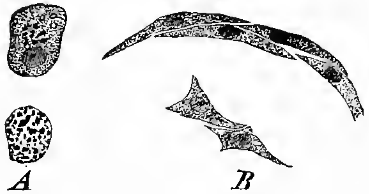

Fig. 360. Atrophic liver-cells.

(× 250)

a simple atrophy with pigmentary deposits
b atrophic liver-cells deformed by compression

Persistent pressure upon the hepatic tissue is likewise very apt to produce progressive atrophy of the parenchyma; the pressure may be from without, as in tight lacing (Art. 215), or from within, as in the case of growing tumours, hydatid cysts, and dilated vessels (Art. 217). Degenerative changes are naturally set up, but these are usually accompanied by simple atrophic wasting of the hepatic cells, the only difference from the senile form being that in the former case the cells are often deformed by the compression (Fig. 360 *B*) before they waste away.

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BOI-TEISSIER: Senile liver *Rev. de méd.* VII **1887**
CZERNY: Retrogressive processes in the liver *A. f. mikrosk. Anat.* XXXV
STATKEWITSCH: Changes in the muscles and glandular tissues in starvation *A. f. exp. Path.* XXXIII **1894**
VOIT: *Hermann's Handb. d. Physiol.* VI Leipzig **1881**

CHAPTER LXVIII

DISORDERS OF THE HEPATIC CIRCULATION

217. **Anaemia** of the liver is either secondary to general anaemia, or the result of local causes. Thus pressure on the liver, or swelling of the liver-cells themselves, may diminish the amount of blood present in the hepatic capillaries. The anaemic tissue is pale, and yellow or brown according to the amount of bile-pigment and of fat contained in the liver-cells.

Congestive hyperaemia of the liver is a very common condition, and is either physiological, as after a meal, or pathological, as in the early stages of inflammation and in affections that determine an increased flow of blood to the intestine. If the congestion is great the volume of the liver may be much increased, and its parenchyma assumes a dark bluish-red or brownish-red colour.

General **venous engorgement**, such as is apt to arise in stenosis and insufficiency of the mitral and tricuspid valves, in weakened conditions of the heart, in emphysema and cirrhosis of the lungs, in right-sided pleuritic effusions, and so on, speedily makes itself felt in the liver, and manifests itself by excessive distension of the hepatic vein and its branches.

When the engorgement is of recent standing the liver appears enlarged and full of blood; and in particular the central parts of the lobules are dark-purple in colour. When it is of older date the liver usually diminishes in size, and its surface is often uneven, granular, or somewhat irregularly knobbed. The cut surface shows a characteristic nutmeg-mottling (**nutmeg-liver**), the central part of each lobule being dark-red and usually a little depressed below the general level of the section, while the periphery (according to the amount of fat present) varies from dark-brown to yellowish-white. When the change has gone still further the livid dark-red portions predominate, and in some parts the lighter tissue may be entirely absent, while the lobules generally are notably diminished in size.

On microscopic examination the intralobular veins and many of the neighbouring capillaries are seen to be dilated (Fig. 361 *a b*). In advanced stages of the disease all the capillaries of the lobules are distended. The hepatic cells which lie between the dilated capillaries are more or less atrophied (*b*), and they are generally beset with yellow or brown pigment-granules, and sometimes also with fat-globules. The degeneration of the liver-cells is always most marked in the central parts of the lobules, and when the disorder of circulation and the dilatation of the capillaries have existed for a long time, some of the cells disappear entirely (*a*), leaving nothing but a few yellowish and yellowish-brown granules and flakes of pigment between the dilated capillaries. The interlobular connective tissue is usually

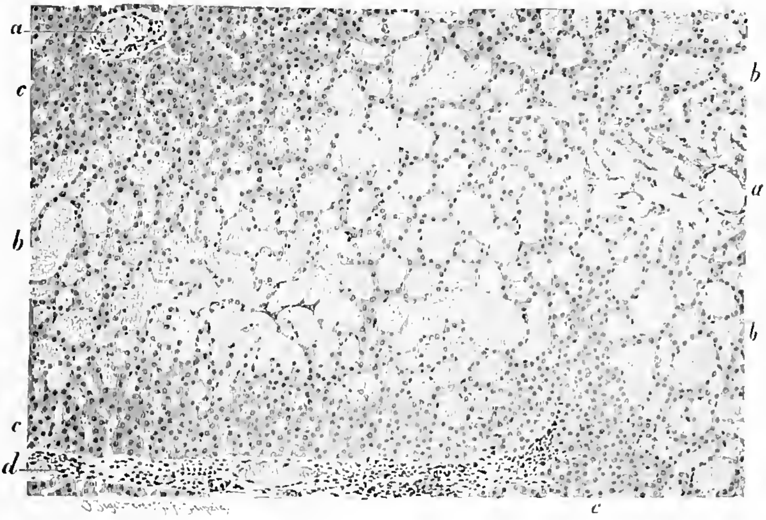

FIG. 361. HEPATIC ATROPHY FROM ENGORGEMENT.

(*Section hardened in Müller's fluid, stained with haematoxylin and eosin, and mounted in Canada balsam :* × 45)

a central vein whose wall and surrounding tissue are somewhat fibroid, delicate fibrous strands passing from it between the dilated capillaries in the spaces vacated by atrophied liver-cells

b median zone of a lobule with dilated capillaries and atrophic columns of liver-cells

c apparently normal outer zone of the lobule

d interlobular connective tissue whose nuclei are somewhat increased in number

unaltered, but now and then it appears to be hyperplastic and infiltrated with small cells (*d*), giving rise to a special form of cirrhosis.

From its chief seat and its mode of origin this affection has been described as central red atrophy, and also as **cyanotic atrophy** or atrophy from engorgement.

Occlusion of the hepatic vessels by thrombosis or embolism, or by endarteritis, induces different morbid changes in different conditions.

Sudden occlusion of the portal vein causes the secretion of bile to cease; but if the obstruction is gradual the secretion goes on. The direct nutrition of the liver-tissue itself is maintained, inasmuch as it is supplied with blood by the hepatic artery. When venous engorgement coexists with portal obstruction, cyanotic atrophy is sometimes induced (KÖHLER).

When the portal vein or its chief branches are gradually but persistently occluded, the arterial channels become dilated and supply the liver with blood sufficient not merely for its own nutrition but also for its functional needs. Only by the obstruction of the smallest of the interlobular (portal) vessels, the blood of which mingles with that of the contiguous arterioles, is the nutrient circulation of the lobules so interrupted or impaired as to cause necrosis or atrophy of the liver-cells. SCHMORL and PRUTZ have recorded cases of eclampsia in which thrombosis of branches of the portal vein was associated with haemorrhagic and anaemic necrosis of the hepatic tissue.

The closure of isolated branches of the hepatic artery has seldom any grave consequences, inasmuch as the branches anastomose freely and by collateral channels maintain the circulation beyond the obstruction. It may happen however, when the blood-pressure within the liver or throughout the body generally is low, that the propelling forces behind the obstruction are insufficient to maintain a continuous flow, and then degeneration and necrosis, with engorgement and even extravasation of blood, are liable to ensue. The haemorrhagic extravasation is however seldom so considerable as to obscure altogether the outlines of the lobules.

The experimental researches of JANSON show that occlusion of the hepatic artery in rabbits is followed by the appearance of patches of necrosis in the liver. The uninjured tissue soon undergoes proliferous changes resulting in some places in the substitution of the necrotic tissue by new connective tissue, while in other parts cysts are produced. Sometimes the biliary ducts in the unaffected region become hyperplastic. In the dog such necroses are not produced.

Haemorrhages within the liver are moreover liable to follow morbid changes in the vessel-walls (as in purpura haemorrhagica and in phosphorus-poisoning), or thrombotic obstruction of the hepatic veins.

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DE BEURMANN and SABOURIN: Hepatic cirrhosis of cardiac origin *Rev. de méd.* VI **1886**

CHARCOT: *Leçons sur les maladies du foie et des reins* Paris **1882**

COHNHEIM: *Vorlesungen über allgemeine Pathologie* Leipzig **1882** (trans. by MCKEE) London **1888–90**

Cohnheim and Litten: Disturbances of the hepatic circulation *V. A.* 67 **1876**

Frerichs: *Klinik der Leberkrankheiten* i Brunswick **1858** (trans. by Murchison) London **1860** and New York **1879**

Gintrac: Obliteration of the portal vein *Journ. méd. de Bordeaux* **1856**

Janson: Changes in the liver after ligation of the hepatic artery *Ziegler's Beiträge* xvii **1895**

Jürgens: Hepatic haemorrhages in eclampsia *Berl. klin. Woch.* **1886**

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Litten: Cirrhosis of the liver *Münch. med. Woch.* **1890**

Moore: Haemorrhage into the liver *Trans. Path. Soc.* xxxvi London **1884**

Murchison: *Diseases of the liver* London **1885**

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Prutz: Condition of the liver in puerperal eclampsia *Inaug. Diss.* Königsberg **1892**

Rattone: Haemorrhagic infarcts of the liver *A. per le scienze med.* xii **1888**

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CHAPTER LXIX

HAEMATOGENOUS INFILTRATION AND DEGENERATION OF THE LIVER.

218. **Haematogenous pigmentation** of the liver is most frequently due to products of disintegration of the red blood-corpuscles, the pigment as a rule consisting of haemosiderin.

The haemosiderin being carried to the liver in the form of granules and small flakes is in the first instance deposited in the capillaries, and is thence taken up by the leucocytes. By and by the cells and with them the pigment-granules and flakes pass

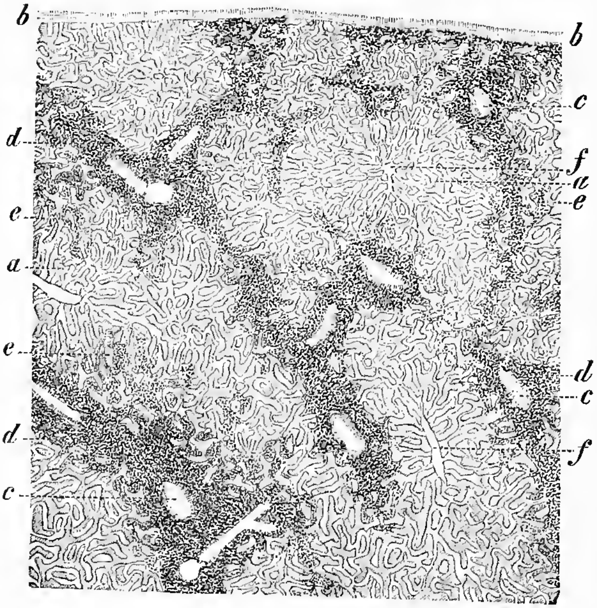

Fig. 362. Haematogenous pigmentary infiltration of the liver.

(*Preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam:* × 20)

a	lobule	*d*	infiltrated interlobular fibrous tissue
b	peritoneum	*e*	pigment in the lobular capillaries
c	portal vessels	*f*	intralobular (central) veins

into the hepatic parenchyma, and thus in the interlobular tissue and in the marginal zone of the lobules the accumulation of haemosiderin may become so considerable that the section appears mottled with rusty-red (Fig. 362 *d e*).

Very frequently haemosiderin is taken up by the tissue-cells themselves, including not only the liver-cells (Fig. 363 *a*) but also Kupffer's stellate cells and the connective-tissue cells of Glisson's capsule, and cases occur (as in malaria or pernicious anaemia) in which the majority of the cells enclose small clusters of yellow granules of haemosiderin, whereby the liver acquires a yellowish-red or rusty appearance.

When the tissue-cells of the liver thus contain haemosiderin. it is probable that it has been for the most part taken up in solution and then precipitated in granular form. But, as experiments with soot and cinnabar have shown, the fixed cells, and in particular the endothelial cells of the blood-vessels and the connective-tissue cells, are also capable of taking solid undissolved particles into their substance: pigmentation by soot has indeed been actually observed in the case of human subjects, the carbonaceous particles having been admitted to the circulation by way of the lungs.

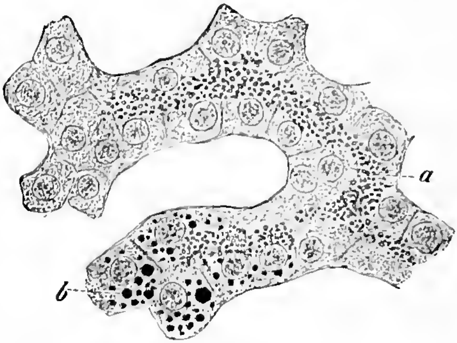

FIG. 363. LIVER CELLS INFILTRATED WITH YELLOW HAEMOSIDERIN-GRANULES FROM A CASE OF PERNICIOUS ANAEMIA.

(Preparation treated with perosmic acid and carmine, and mounted in glycerine : × 250)

a pigment-granules
b cells undergoing fatty degeneration

If the blood contains, as in leukaemia, an excess of colourless corpuscles, these are in like manner especially apt to be deposited in the liver, and give rise to what is called **leukaemic infiltration**. Multiplication of the leucocytes within the liver seems also to take place, with the result that the organ becomes much enlarged, and the lobules appear separated from each other by a broad zone of greyish-white tissue. Sometimes this general or diffuse infiltration is accompanied by nodular aggregations of cells, within which the connective tissue is looser and less compact, so that the nodules assume the appearance of lymphadenoid tissue.

Noxious particulate matters, like microbes, may be deposited in the liver in the same way as innocuous substances, and give rise to necrosis and inflammation of the liver-substance (Art. 223).

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Arnold: *Untersuchungen über Staubinhalation* Leipzig **1885**
Arnstein: Melanaemia and melanosis *V. A.* 61 **1874**
Asch: The deposition of fat and pigment in the stellate cells of the liver *Inaug. Diss.* Bonn **1884**
Bignami: Chronic malaria *Cent. f. allg. Path.* **1894**
Bunge: The iron contained in the liver *Z. f. physiol. Chem.* XVII **1892**
de Filippi: Researches on 'ferratin' *Ziegler's Beiträge* XVI **1894**
Frerichs: *Klinik der Leberkrankheiten* I and II Brunswick **1858–1861** (trans. by Murchison) London **1860–62**
Guarneri: Alterations of the liver in malaria *Atti della reale Accad. di Roma* (year XIII) 2nd series III Rome **1887**
Hindenlang: Pigmentary infiltration in haemorrhagic purpura *V. A.* 79 **1880**
Hoffmann and Langerhans: Deposits of cinnabar *V. A.* 48 **1869**
Peters: Deposits of iron in the organs in various diseases *Inaug. Diss.* Kiel **1881**, and *D. A. f. klin.* XXXII **1882**
Ponfick: The fate of granular colouring-matters in the organism *V. A.* 48 **1869**
Quincke: Pathology of the blood (siderosis) *D. A. f. klin. Med.* XXV XXVII **1880** and XXXIII **1883**
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Rothe, P.: The stellate cells of the liver *Inaug. Diss.* Munich **1882**
Schmidt: Physiology of the liver *Biol. Centralbl.* X **1890**
Soyka: Coal-dust metastasis *Prager med. Woch.* **1878**
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Welch: Cirrhosis hepatis anthracotica *Johns Hopkins Hosp. Bull.* II Baltimore **1891**

219. The liver is one of the organs of the body that serve for the storage of fat, and so nearly always contains a certain proportion of it. When the quantity is small it does not affect the colour of the liver; when it is larger, the yellowish-white tint of the fat blends with the normal purplish or brownish-red hue of the liver, and the organ accordingly becomes yellowish-brown, or when the fat is abundant and the amount of blood present is small it appears of a pale yellowish-white tint.

The **fatty liver**, distinguished by its containing an excessive proportion of deposited fat, is large, and when cold is somewhat firm to the touch. Globules of oil are readily expressed by the knife-blade from the yellowish surface of section. The individual lobules appear enlarged.

The fat lies in the form of large drops, principally in the interior of the liver-cells (Figs. 364 and 366 *a b*), but the stellate cells may also contain a certain amount. The deposit of fat first takes place in the peripheral portions of the lobules (Fig. 364 *b*), and thence extends to the more central parts. In cases where the margins are completely infiltrated with fat, while the centre is still free from it, and therefore brown or reddish-brown in colour, the contrasted tints produce a characteristic mottling of the surface of section which has given rise to the term **fatty nutmeg-liver** (Fig. 364).

The fat in cases of fatty infiltration of the liver is brought to the organ ready-formed, having been either ingested as food, or elaborated in some other part of the body from albumen or carbohydrates. Its accumulation in the liver must be due to excessive ingestion or production on the one hand, or on the other to defective metabolism of that which is normally present in the body. It is owing to the latter cause that we so frequently find fatty livers in patients who have died of pulmonary tuberculosis.

The fat in the liver-cells is deposited at first in small globules (Fig. 366 *a*); but these soon coalesce into larger drops (*b*), until at length the cell is transformed into a spherule of oil.

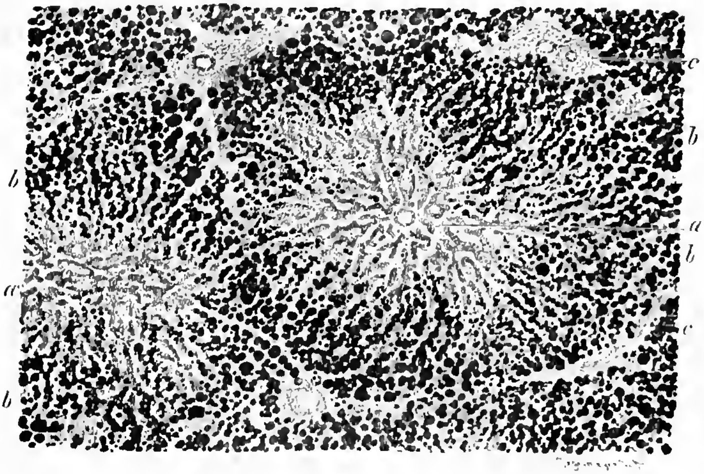

FIG. 364. FATTY LIVER FROM A MAN WITH PULMONARY TUBERCULOSIS.
(*Preparation fixed in Flemming's acid solution, and stained with saffranin*: × 30)
a central portions of the lobules *b* peripheral fatty zone
c interlobular connective tissue

220. The deposits of **amyloid substance**, which produce what is known as the lardaceous, waxy, or **amyloid liver**, are associated with general disorders of nutrition, such as result from chronic tuberculosis, chronic suppuration, and syphilis. They give rise in well-marked cases to induration and enlargement of the liver, the degenerate tissue having a peculiar semi-translucent appearance like the rind of boiled bacon. The unaltered tissue is yellow, brown, or reddish-brown in colour, according to the amount of fat present in the liver-cells and the quantity of blood contained in the capillaries.

The amyloid deposit is found mainly in the middle zone of the lobules (Fig. 365 *b*), the central and peripheral zones remaining free; but the whole of the lobule may eventually become affected. The primary seat of the change is in the outer walls of the capillaries, the amyloid substance being deposited in the form of hyaline masses between the endothelium of the capillaries and the trabeculae of liver-cells (Fig. 365 *b*). Sometimes the vessels running in Glisson's capsule (*c*) are also affected by the degeneration. The liver-cells rarely become amyloid themselves, but as the amyloid deposits around them increase in bulk they become compressed, and some of them undergo fatty degeneration. Not infrequently an amyloid liver is also morbidly altered by tuberculous or syphilitic disease.

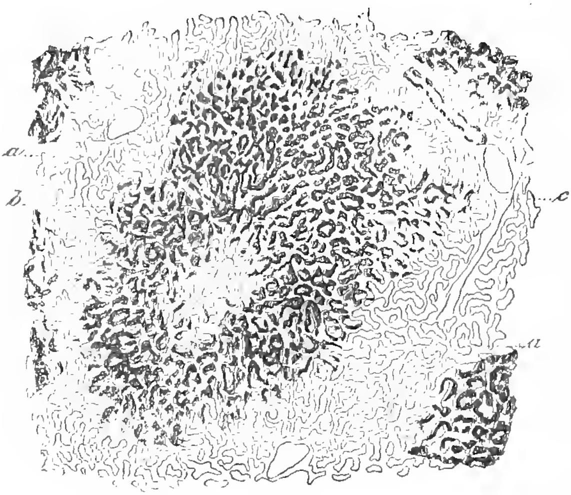

Fig. 365. Section of amyloid liver treated with iodine (× 35).

a normal hepatic tissue *b* amyloid tissue *c* capsule of Glisson

In rare cases the degeneration is not generally diffused, but takes the form of circumscribed amyloid deposits in some of the hepatic vessels, or of nodular aggregations in the interlobular connective tissue.

References on Amyloid Liver.

Birch-Hirschfeld: Condition of the liver-cells in the amyloid liver *Wagner's Festschrift* Leipzig **1887**
Böttcher: Amyloid degeneration of the liver *V. A.* 72 **1878** and 84 **1881**
Heschl: Idem *Wiener Sitzungsber.* lxxiv **1877**
Schüppel: Lardaceous liver *Ziemssen's Cyclop.* ix New York **1880**
Schütte: Idem *Inaug. Diss.* Bonn **1877** (with good illustrations)
Tilssen: Researches on amyloid liver *Inaug. Diss.* Königsberg **1877**
Wichmann: Amyloid degeneration *Ziegler's Beiträge* xiii **1893**

221. **Haematogenous degenerations** of the liver-cells most frequently take the form of necrosis, fatty degeneration (Fig. 366 *c d e f*), or cloudy swelling (Fig. 367); but vacuolation, dropsical degeneration, colloid change (Mattei, Taruffi), and pigmentary degeneration are also observed (Arts. 216–218).

Fatty degeneration implies the formation of fat from the albuminous protoplasm of the liver-cells themselves, the process sometimes resulting in their complete destruction and disintegration (Fig. 366 *e f*). When it is sufficiently advanced the cells appear turbid, yellow, and swollen, and the liver assumes a dull greyish or greyish-yellow colour.

The haematogenous degenerations are for the most part due to infective and toxic agencies; but persistent pyrexia, general anaemia, and disorders of the circulation are capable of inducing degenerative changes, of which fatty degeneration (Fig. 363 *b*) and necrosis are the commonest varieties.

Of poisonous substances, phosphorus, arsenic, antimony, and many poisonous mushrooms give rise to extreme fatty degeneration, even to the extent of complete destruction of the cells, and in the case of phosphorus-poisoning this may be accompanied by haemorrhage. Among the infective diseases that induce cloudy swelling and fatty degeneration are typhoid fever, relapsing fever, small-pox, scarlet fever, septicaemia, and yellow fever. Necrosis is generally associated with the invasion of pyogenic micrococci, the death of the cells being usually followed by suppurative inflammation (Art. 223).

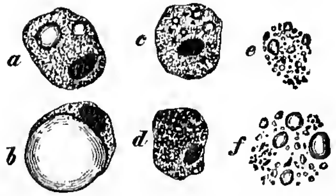

FIG. 366. FATTY INFILTRATION AND FATTY DEGENERATION OF LIVER-CELLS.

(*Fresh preparation*: × 400)

a b fatty deposit in the cells from a case of fatty liver
c d e f fatty degeneration and disintegration of the cells

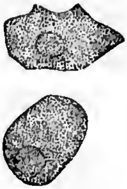

FIG. 367. GRANULAR CLOUDY SWELLING OF THE LIVER-CELLS.

(*Cells scraped from the liver of a man who died of septicaemia; floated in salt-solution*: × 350)

SCHMORL and PRUTZ have described cases of ischaemic and haemorrhagic necrosis in fatal puerperal eclampsia. STRAUS, ROUX, HANOT, GILBERT, DOYEN, and others state that the liver in fatal cases of cholera contains greyish dirty-yellow patches within which the cells are swollen and will not stain with the usual reagents. In small-pox the liver-tissue is sometimes beset with minute necrotic foci (WEIGERT, BOWEN), and thus undergoes a form of inflammatory infiltration. In typhoid fever we find, over and above the general or diffuse degeneration, a number of small necrotic patches, together with foci of cellular infiltration about the vessels, and circumscribed aggregations of proliferous cells that look like lymph-follicles: in these patches and cellular clusters the characteristic bacilli have been detected (GAFFKY, FRÄNKEL, E. SIMMONDS, CYGNAEUS). Malaria gives rise not only to pigmentary but also to degeneration of the cells and disturbances of the hepatic circulation (Bignami). Cellular degeneration and necrosis, and inflammatory changes (Nauwerck), have also been demonstrated in the liver in cases of the acute febrile form of jaundice which has been called 'Weil's disease' or 'bilious typhoid.'

When haematogenous degeneration in its earlier or later stages is associated with exudation and proliferation, it acquires the character of an inflammation, and may be described as a **haematogenous hepatitis.** When degeneration of the essential elements (the liver-cells) is the predominant feature, the affection is termed **parenchymatous hepatitis.** But if the cellular infiltration of the interlobular connective tissue is more prominent than the degenerative changes the process is regarded as an **interstitial hepatitis.**

The milder forms of degeneration and hepatitis are capable of recovery, by restoration or replacement of the degenerate cells, without leaving any permanent change behind. The graver forms give rise, however, to persistent structural alterations, such as local defects of the hepatic parenchyma and hyperplasia of the connective tissue ; in many cases these are accompanied by morbid proliferation of the biliary ducts and liver-cells (Arts. 222–226).

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Aufrecht : Phosphorus-poisoning *D. A. f. klin. Med.* xxiii **1879**
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Fränkel : Phosphorus-poisoning *Berl. klin. Woch.* **1878**
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222. **Acute yellow atrophy**, a disease classed with the haematogenous degenerations of the liver, is a very grave affection, characterised by a great and sudden diminution of the size of the organ. Within a few days, or in from one to two weeks, the liver may lose as much as half its bulk or more. The disease makes its appearance either in patients suffering from recognised infective disorders, such as traumatic or puerperal septicaemia and syphilis, or in persons that were previously healthy. It is very probably an effect of various forms of toxaemia induced by more than one kind of infection. In several instances bacteria have been found in the liver; and it is possible that in particular cases the efficient poison has been absorbed from the intestinal tract. Phosphorus-poisoning gives rise to degenerative changes that somewhat resemble those of acute yellow atrophy.

The onset of the disease cannot as a rule be recognised with certainty, and it is not likely to be diagnosed until, after various indefinite prodromata, grave symptoms like vomiting, intense headache, mental excitement, delirium, convulsions, and coma supervene in association with jaundice.

The degeneration in acute yellow atrophy consists in a fatty disintegration (Fig. 366 *e f*) of the albuminoid constituents of the liver-cells, beginning at the periphery and rapidly advancing toward the centre of the lobules. The degeneration is at times accompanied by serous exudation from the vessels and emigration of leucocytes. The epithelial cells of the radicles of the biliary ducts (or canaliculi) also become fatty. The diminution in the size of the liver is due to the rapid liquefaction and absorption of the products of disintegration.

The *post-mortem* appearance of the liver varies with the stage at which death occurs. When the fatty change has just taken place the liver is of an ochreous yellow colour (hence the term yellow atrophy); but after the detritus has become liquefied and absorbed its tissue becomes grey, or if much blood be present greyish-red or red (**red atrophy**).

Uniformly diffused fatty degeneration causes the outlines of the lobules to be obscured. If the degeneration is unevenly diffused, the blood irregularly distributed, and the absorption of detritus not uniform in the different parts, the liver has a mottled or variegated appearance on section. The presence of yellow pigment-granules may modify the coloration. The tissue itself is always soft and flabby. The lobules are smaller than normal.

If the disease is not fatal until between the second and the fourth week after the onset of the graver symptoms, the contours of the lobules are in some places traceable, in others obscured; while the cut surface is mottled with brownish-red, reddish-yellow, and brownish-yellow, and occasionally over a considerable extent the tint is uniform, or merely spotted.

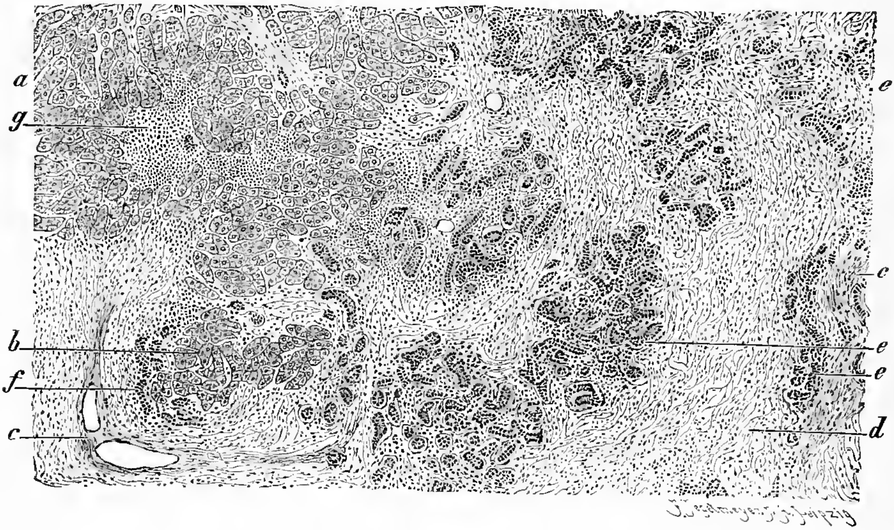

Fig. 368. Acute yellow atrophy in the fourth week of the disease.
(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin*: × 30) ·

a uninjured liver-tissue
b surviving remnants of hepatic trabeculae
c Glisson's capsule
d region in which the liver-cells have been destroyed and the connective tissue is proliferous
e f newly-formed epithelial columns and tubules
g hyperplastic cellular connective tissue

In the greatly-altered portions the trabeculae of liver-cells may be completely absent (Fig. 368 *d*), and in such regions nothing remains but connective tissue in process of active proliferation. In other regions that are less affected, we find irregular islands of liver-tissue (*a b*) that have escaped destruction, and either are still fatty or have already regained their normal appearance. In the later stages the hyperplastic fibrous tissue invariably contains large numbers of slender cellular columns and tubules resembling small biliary ducts (*e*). So far as can be made out from histological examination, these proliferous changes start partly from the biliary ducts and partly from surviving hepatic trabeculae; they are apparently the outcome of an effort to reproduce the lost hepatic parenchyma.

When the degeneration is not extreme and extensive, recovery may take place by regenerative proliferation. The severer types, when not fatal, leave behind them textural changes of the nature of cirrhosis, particularly in cases where strands of connective tissue containing newly-formed biliary ducts are formed between the islands of surviving and hyperplastic liver-tissue.

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CHAPTER LXX

HAEMATOGENOUS INFLAMMATIONS

223. **Haematogenous suppurative hepatitis** is usually due to the conveyance of pyogenic micrococci to the liver by way of the portal vein or hepatic artery. In rare cases an infective embolus passing from the superior vena cava against the blood-stream furnishes the starting-point. In new-born infants the umbilical veins sometimes convey infection to the liver. In the majority of cases the ordinary pyogenic micrococci are the exciting cause, but other bacteria, such as the *Bacillus coli communis*, may give rise to suppuration in the liver: in the forms associated with antecedent dysentery the specific amoeba of that disease seems to play a part; and at any rate this organism has been found in hepatic abscesses.

Haematogenous suppurative hepatitis very often presents the characters of a metastatic inflammation, in connexion with suppuration of other organs or parts, such as the limbs or head, or with grave forms of intestinal inflammation like dysentery. In other cases the hepatic affection is the first local manifestation of the infection, cases of primary suppurative hepatitis of this kind being frequently observed in tropical countries. But it should be noted that in all probability these tropical abscesses originate as a rule in the bile-ducts, and are thus to be classed with the biliary affections (Art. 225).

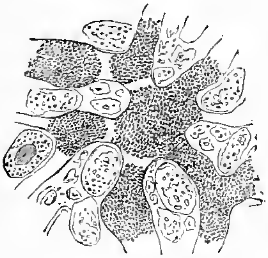

FIG. 369. COLONIES OF MICROCOCCI WITHIN THE HEPATIC CAPILLARIES.

(Section hardened in alcohol, stained with Bismarck-brown, and mounted in Canada balsam)

The micrococci of pyaemia that form zoogloea-clusters, when they enter the liver along with the blood, lodge first in the capillaries (Fig. 369 and Fig. 370 *c*), and then in the smaller venules. There they form colonies which more or less fill up and distend the lumen of the vessel. At first the adjacent liver-tissue appears unaltered, but after a time the cells become turbid and swollen, lose their nuclei, and break up into fragments of various sizes (Fig. 369).

The bacterial colonies spread more and more widely through the vessels, while new colonies are at the same time formed, so that soon a great number of affected lobules have their capillaries (Fig. 370 *c*) and often their intralobular veins crammed with bacteria, while at the same time the necrosis of the liver-cells advances steadily (*b*).

These changes are soon accompanied by intense inflammation, which proceeds both from the interlobular capillaries (*d*) and from the central venules (*e*), and is indicated by dense cellular infiltration of the adjacent tissue. This is the first stage in the formation of an abscess. Soon the liquid and cellular exudations become more abundant, and the necrotic cells break up and liquefy. The purulent infiltration is thus converted into a **hepatic abscess.** This is in brief the general course of the process, but it may of course be modified in numerous ways.

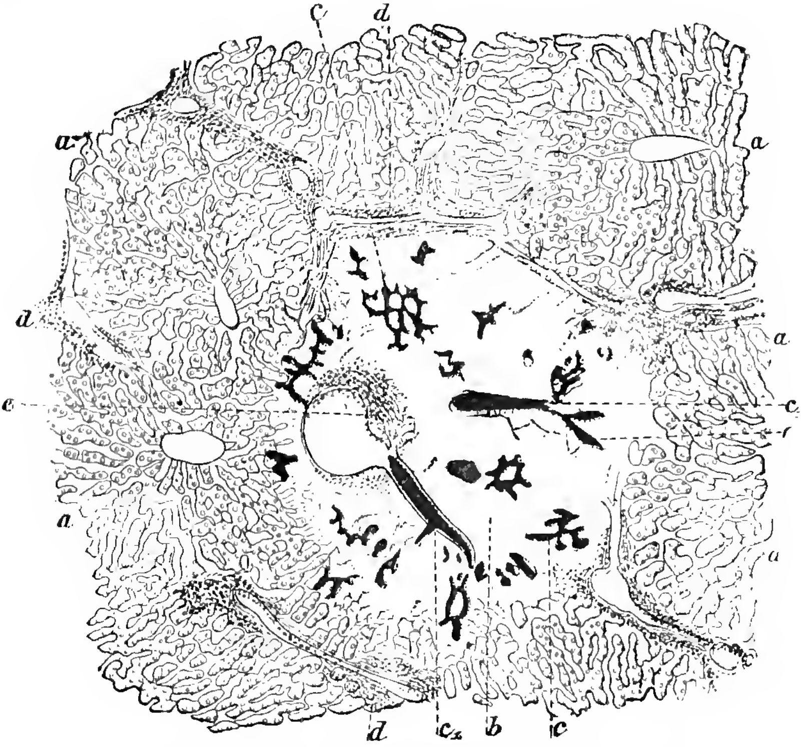

FIG. 370. METASTATIC COLONIES OF MICROCOCCI IN THE LIVER.

(*Preparation stained by Gram's method with gentian-violet and vesuvin, and mounted in Canada balsam:* × 40)

a normal lobules
b necrotic lobules
c capillaries and veins (c_1) filled with micrococci
d small-celled infiltration of the interlobular tissue
e aggregation of round-cells within and about a vein, into which opens a central vein (c_1) crammed with micrococci

As necrosis supervenes the tissue of the lobules becomes of a greyish or greyish-yellow colour. Then the parts that are on the point of suppurating become gradually yellow or yellowish-white, and presently the whole of the affected patch undergoes liquefaction, till at length its place is occupied by pus which is either liquid throughout or mingled with discoloured shreds of necrotic tissue. The surrounding parts are likewise discoloured, infiltrated with pus, and in process of liquefaction.

The abscesses may be single or multiple. The other parts of the parenchyma show a greater or less degree of turbid swelling, sometimes accompanied by extravasations of blood which become slaty-grey in colour when putrefaction sets in.

Large abscesses may occupy an entire lobe. Minute multiple abscesses sometimes coalesce into larger ones. If an abscess is situated directly underneath the serous membrane, more or less intense inflammation of the latter is the result.

In many cases abscess of the liver, or the injury to which it is secondary, brings about the death of the patient. But when death does not take place granulation-tissue is developed around the abscess-cavity and forms for the rest of the organ a kind of protecting membrane. Small abscesses may disappear, their contents being entirely absorbed; and a cicatrix varying in size with the size of the abscess is left. Larger abscesses become notably contracted by absorption and inspissation of the pus. The inspissated pus is always enclosed by a tough and thickened fibrous wall, and sometimes becomes calcified.

Abscesses frequently break into surrounding parts. This issue is the most favourable when adhesions have been set up between the liver and the abdominal wall, the diaphragm and lung, or the intestine; and the pus is then evacuated through the adherent parts to the outside of the body, into the bowel, or into one of the bronchi. Rupture into the cavity of the pleura, the pericardium, or the peritoneum is more dangerous. In the latter case the inflammation of the corresponding serous membrane will be local or general according as previous adhesions between the liver and the other viscera are present or absent.

Biliary suppurations are dealt with in Art. 225.

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224. Under the term **chronic haematogenous hepatitis** is comprised a group of diseases of the liver that are characterised by hyperplasia of the connective tissue, cellular infiltration, and destruction of the hepatic parenchyma, generally accompanied by regenerative production of new biliary ducts and liver-cells, and by morbid pigmentation.

The causes of chronic hepatitis are still imperfectly understood, but there is no doubt that the disease may be due to various noxious agents that either are introduced from without, or are formed in the intestine or in the tissues of the body itself, and then reach the liver by way of the blood. There is good reason for thinking that in a certain proportion of cases the excessive use of alcohol is the exciting cause, but it would be a mistake to assume that this accounts even for the majority of cases. Of known infective diseases syphilis is the one which is most apt to induce chronic hepatitis. It is probable, however, that other forms of infection give rise to morbid changes which are regarded as varieties of chronic hepatitis, and that some of the affections so described are in part the results of certain acute degenerations (Art. 222).

The first stage of the disease may apparently be indicated by degeneration of the liver-cells as well as by inflammatory exudation and fibrous hyperplasia, this view being supported by the fact that in cases of acute infection and acute poisoning sometimes the glandular parenchyma, and sometimes the connective tissue of the vessels, is primarily and chiefly affected. It is moreover highly probable that what we call cirrhosis of the liver may in some cases be the terminal stage of an infective or toxaemic affection that began acutely.

The terminal stage of the disease is either a condition of induration with contraction, which is termed **atrophic cirrhosis** or simply cirrhosis (in the stricter sense of the term), or of induration without contraction and very frequently indeed with actual enlargement of the liver, to which the term **hypertrophic cirrhosis** or hypertrophic induration is applied.

In atrophic cirrhosis, also known as Laennec's cirrhosis, the liver is more or less reduced in size, in some cases weighing not more than 700 to 900 grammes. Its surface is either finely or coarsely granulated (Fig. 371), or divided into irregular lobes by shrunken cicatricial bands (Fig. 372). From the external appearance of the organ it is accordingly described as a granular, nodular, 'hob-nailed,' or lobulated cirrhotic liver.

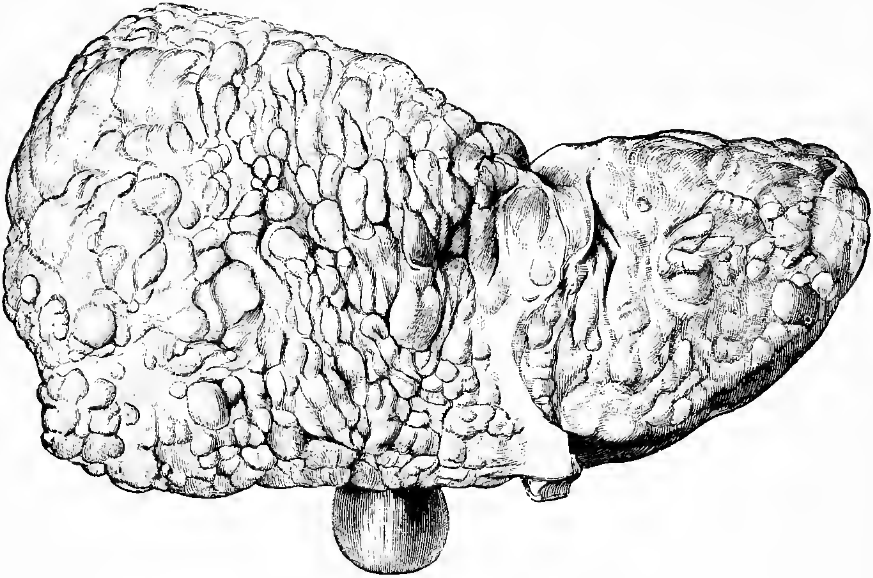

FIG. 371. ATROPHIC CIRRHOSIS OF THE LIVER.
(*One-half the natural size*)

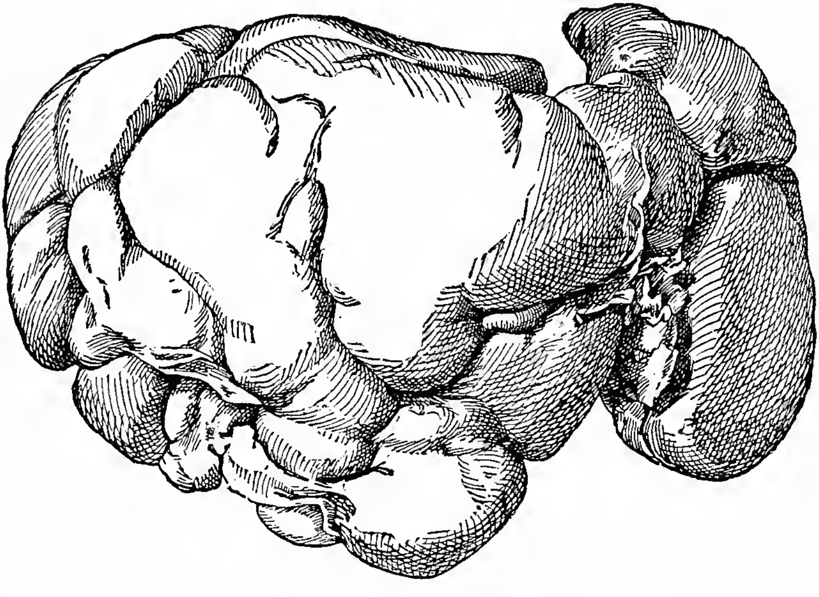

FIG. 372. LOBULATED LIVER.
(*Upper and anterior aspect: one-half the natural size*)

The colour of the firm resistent tissue, which creaks when cut with the knife, may be yellowish, yellowish-brown, yellowish-green, yellowish-red, or mottled. On careful inspection of the cut surface the parenchyma is generally seen to be traversed by bands of connective tissue (Fig. 373 *b*), which in the case of granular cirrhosis enclose small islands (*a*), and in the nodular and lobulated forms larger tracts of hepatic tissue (annular cirrhosis): these fibrous bands frequently start from the cicatricial depressions on the surface of the liver.

An indurated liver that is of normal size or enlarged has in some instances a smooth surface, in others it is slightly granular or nodular.

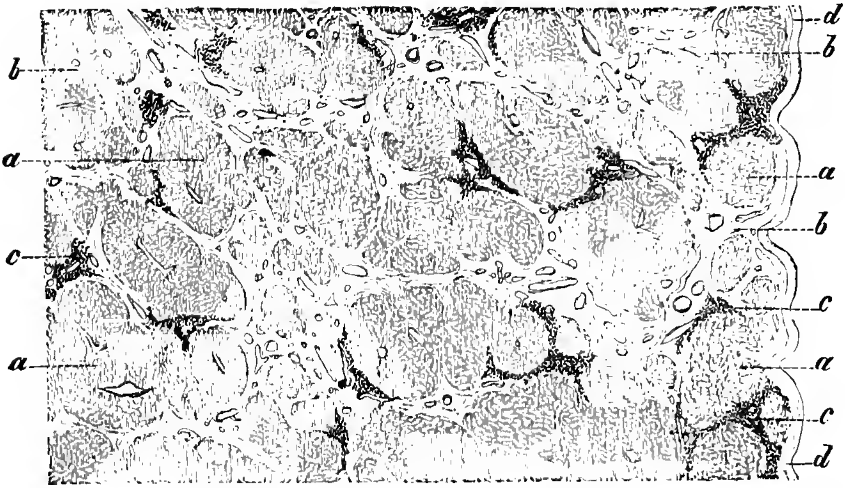

FIG. 373. ATROPHIC CIRRHOSIS OF THE LIVER.

(Preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam: × 8)

a islands of hepatic tissue
b bands of vascular fibrous tissue
c cellular infiltration
d thickened peritoneal covering

When the enlargement is considerable the weight of the liver may reach 3000 to 4000 grammes. The colour of this form as of the other varies in different cases, being at times brownish-yellow, at times rusty-red or yellowish-green, at others mottled with diverse tints. On the surface of section reticulate bands of fibrous tissue are often visible; but varieties are met with in which the texture appears perfectly uniform, neither the lobules nor the interlobular fibrous strands being recognisable.

The fibro-cellular hyperplasia affects mainly the interlobular connective tissue (Figs. 374 *b* and 375 *b e*), but may extend along the capillaries to the intralobular tissue (Fig. 376 *d e f*). When the morbid change is not far advanced the new fibrous tissue is seen to be in some parts fully developed (Fig. 375 *b* and Fig. 376 *f*), in others it is still in the stage of granulation-tissue, consisting of proliferous connective-tissue cells and leucocytes (Fig. 374 *b*, Fig. 375 *e*, and Fig. 376 *d e*) with newly-formed vessels (Fig. 376 *g*). If the hyperplastic process has come to a standstill, the cellular granulation-tissue can no longer be traced.

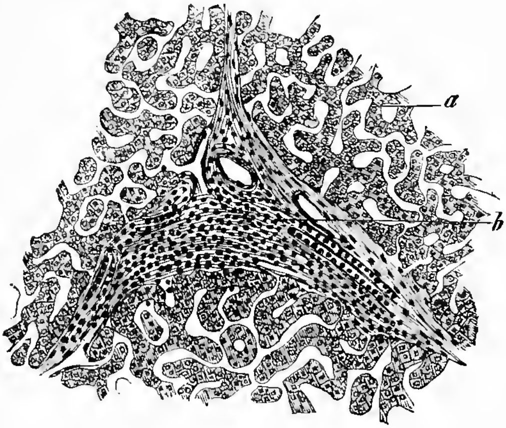

FIG. 374. RECENT INTERSTITIAL HEPATITIS.

(*Preparation hardened in Müller's fluid and alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 80)

a normal liver-tissue
b interlobular connective tissue infiltrated with leucocytes

When the liver comes under examination the hepatic cells are in many cases still unchanged, even though the hyperplastic fibrous tissue surrounds the trabeculae (Fig. 375 *a*) or penetrates between them (Fig. 376 *d e f g*). In other cases the cells appear to be proliferous, especially when the liver is enlarged, or when it is studded with projecting islands of hepatic tissue. Instances also occur in which the cells have here and there undergone atrophy and fatty degeneration, so that the persisting portions of the parenchyma have the appearance characteristic of fatty liver, and the condition might be described as one of **fatty cirrhosis.**

The degenerative changes in the liver-cells may be directly due to the original injurious agency affecting the liver, or they may be secondary to disturbances of circulation and nutrition induced by the fibrous hyperplasia; but it is to be noted that even in cases where the overgrowth of fibrous tissue is abundant and wide-spread the liver-cells often suffer but little. The proliferous changes in the liver-cells are probably to be regarded as reparative and compensatory, but it is not impossible that the presence of irritating matters in the blood may stimulate the cells to proliferation.

The **biliary ducts** in most cases show signs of proliferous multiplication, leading to the formation of numerous canalised columns of cells and gland-like tubules, which permeate the hyperplastic interlobular connective tissue (Fig. 375 *d*); but cases occur in which this multiplication of the ducts is scanty or entirely absent.

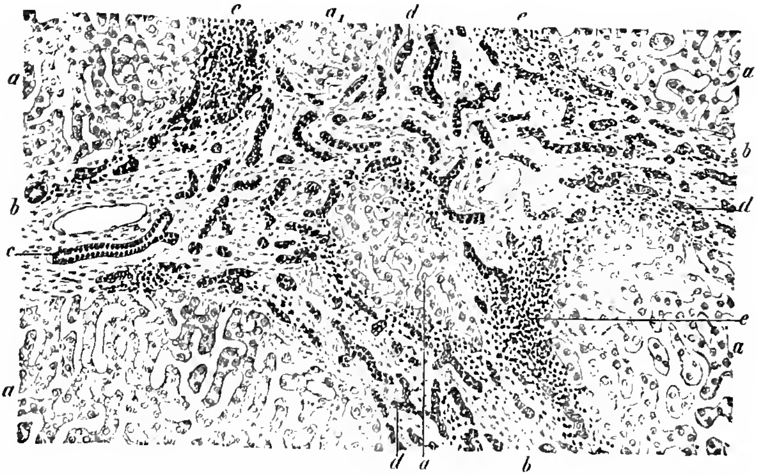

FIG. 375. FIBROUS HYPERPLASIA AND PROLIFEROUS BILIARY DUCTS IN CHRONIC HEPATITIS.

(*Preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam* : × 60)

a a_1	hepatic lobules
b	hyperplastic connective tissue
c	old biliary ducts
d	newly-formed biliary ducts
e	cellular infiltrations

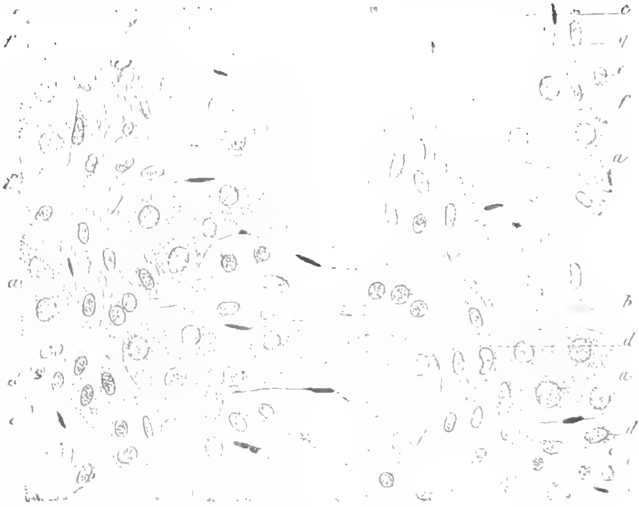

FIG. 376. INTRAVASCULAR DEVELOPMENT OF CONNECTIVE TISSUE IN HYPERTROPHIC CIRRHOSIS.

(*Preparation hardened in alcohol, stained with picro-carmine, and mounted in Canada balsam* : × 300)

a	hepatic trabeculae
b	wall of capillary
c	nuclei of capillary wall
d	formative cells (fibroblasts)
e	leucocytes
f	fibrillar connective tissue with cells of various forms
g	newly-formed capillary penetrating from the interlobular portal sheath into the granulation-tissue

Many of the **hepatic blood-vessels** become obliterated and disappear, and many of the branches of the portal vein become impervious, leading to extreme portal engorgement and consequent ascites. Associated with this there is often marked dilatation of the vessels connecting the gastric and haemorrhoidal veins with the oesophageal, lumbar, and spermatic veins; and occasionally dilatation of the persistent channels of the umbilical (BAUMGARTEN) and parumbilical veins, and of the subserous veins of the round ligament, leads by degrees to dilatation of the subcutaneous veins of the abdominal wall, which then form a tortuous plexus or *caput medusae*. Cases of both the hypertrophic and the atrophic forms of cirrhosis have however been recorded in which there was no perceptible disturbance of the portal circulation and no ascites.

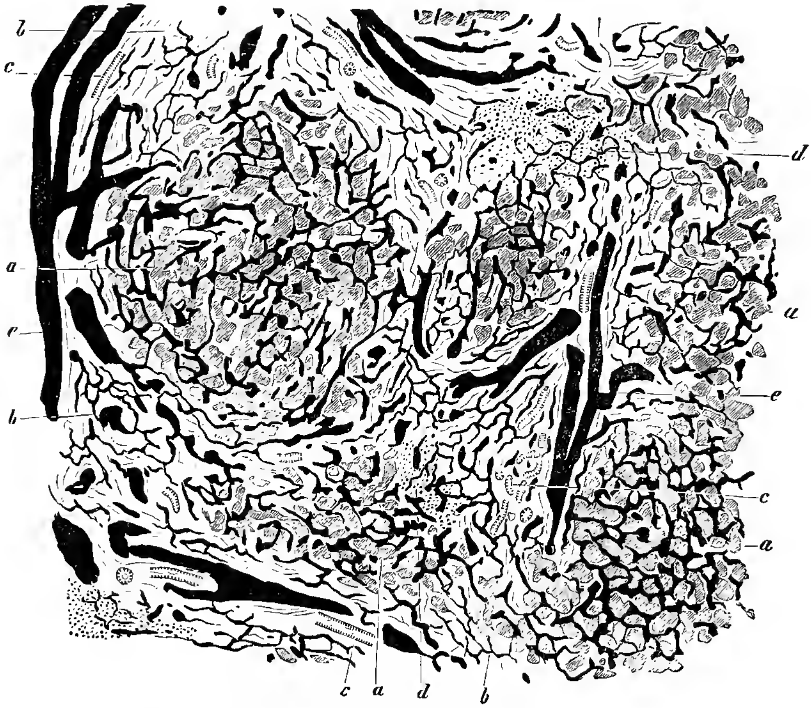

FIG. 377. GRANULAR ATROPHIC CIRRHOSIS OF THE LIVER.

(*Vessels injected with blue gelatine through the hepatic artery; section hardened in alcohol, and stained with carmine*: × 25)

a remnants of liver-tissue
b newly-formed fibrous tissue
c biliary ducts
d small-celled infiltration
e interlobular (portal) veins

On the other hand a large number of new capillary vessels are generally formed in the liver, and these derive their blood mainly from the branches of the hepatic artery. On this account it is usually difficult to inject the vessels of a cirrhotic liver through the portal vein, while a perfect injection can be obtained through the hepatic artery (Fig. 377).

Pigmentary deposits are common in the cirrhotic liver, and give rise to a yellowish, yellowish-brown, reddish-yellow, yellowish-green, or olive coloration. The pigment consists of yellowish, reddish-brown, or reddish-yellow granules and flakes, which lie partly in the hyperplastic fibrous tissue, partly in the liver-cells and in Kupffer's stellate cells, as well as in the endothelium of the vessels.

In some cases the pigment consists chiefly of haemosiderin; but there are forms of chronic hepatitis in which the hyperplastic connective tissue and the liver-cells contain large quantities of iron in granules and flaky masses, lying for the most part in the peripheral zone of the lobules. It is natural to assume that in these instances the disease is associated with excessive blood-destruction.

In other cases the colouring matter consists mainly of bile-pigment, and is deposited chiefly in the liver-cells, while the fibrous tissue contains few if any pigment-granules. This form of pigmentation is probably referable to the obstruction or occlusion of the biliary channels brought about by the morbid changes in the interlobular fibrous tissue. The outflow of bile from the liver is thereby hindered, and the retained bile stagnates in the ducts. This explanation is supported by the fact that in a number of cases cirrhosis and induration of the liver are associated with **jaundice**.

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CHAPTER LXXI

BILIARY INFILTRATION AND INFLAMMATION

225. When the outflow of bile from the liver is hindered in any way, as by occlusion or compression of the common bile-duct or its branches, the organ becomes stained with bile, and assumes a yellowish-green or olive-green colour; this staining has been termed icterus or jaundice of the liver.

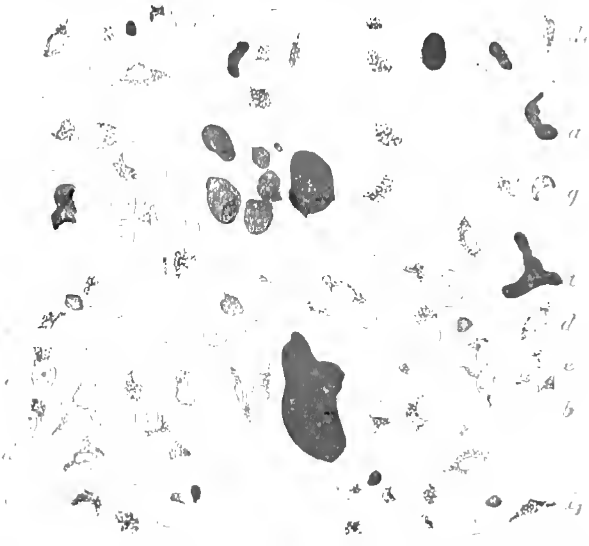

FIG. 378. JAUNDICE OF THE LIVER FROM RETENTION OF BILE DUE TO COMPRESSION OF THE COMMON DUCT BY CANCER OF THE GALL-BLADDER.

(*Preparation hardened in corrosive sublimate, stained with alum-carmine, and mounted in Canada balsam*: × 365)

a	intralobular ducts somewhat distended with bile	*d* d_1	Endothelial cells stained with bile and Kupffer's stellate cells
b	large flake of bile-pigment in a greatly dilated intralobular duct	*e*	loose pigmented endothelial cells
c	bile-pigment in the liver-cells	*f*	pigment-masses enclosed in cells
		g	rupture into a capillary of a pigment-mass contained in a biliary duct

The retained bile-pigment is in the first instance deposited in the biliary capillaries (Fig. 378 *a b*) and the liver-cells (*c*), in the former as yellow granules containing no iron, in the latter as homogeneous yellow flakes that become green when the tissue is treated with corrosive sublimate. When the retention is of long duration, the intralobular biliary capillaries are sometimes greatly distended (*b*).

Kupffer's stellate cells and the endothelial cells ($d\ d_1\ e$) of the vessels are stained in like manner, and the pigmented endothelial cells are occasionally detached from their substratum (*e*).

The liver may tolerate this biliary pigmentation for weeks or even months without undergoing any noteworthy changes; but it not infrequently happens that in the course of time necrotic foci make their appearance in the lobules, the affected liver-cells losing their nuclei and being converted into shapeless masses. Moreover, interlobular and intralobular cellular infiltration and fibrous hyperplasia are apt to be set up, and at length even suppurative inflammation (Fig. 379 *c d*) may supervene, the suppuration involving primarily the interlobular ducts and the tissue about them or the interior of the lobules (*d*).

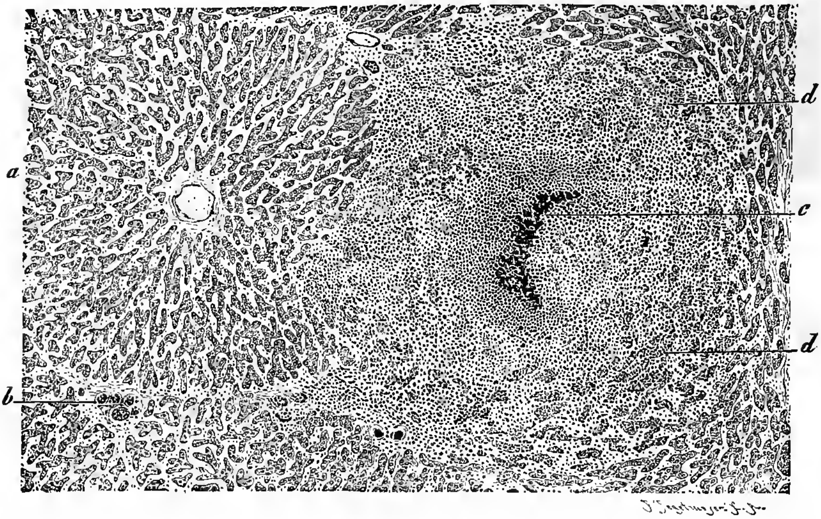

FIG. 379. SUPPURATIVE BILIARY HEPATITIS FROM COMPRESSION OF THE COMMON DUCT AND INVASION OF THE BILE-CHANNELS BY THE BACILLUS COLI COMMUNIS.

(*Preparation hardened in alcohol, and stained with haematoxylin and eosin:* × 45)

a hepatic lobule in transverse section
b Glisson's capsule with bile-ducts
c collection of pus with bile-stained masses of necrotic detritus
d necrotic liver-tissue infiltrated with cells

Chronic retention and stagnation of the bile may furthermore lead to degeneration and inflammation of the liver, in other words to **biliary hepatitis**. This condition is equally liable to supervene whether the obstruction is in the common duct outside the liver, or within it in the interlobular channels. It is thus possible for an originally haematogenous hepatitis, giving rise to retention of bile, to become at length complicated by a secondary biliary hepatitis, and so to assume a rapidly progressive character.

The causes which determine whether retention of bile shall be long tolerated without appreciable consequences, or shall after a comparatively short period give rise to hepatitis, cannot in all cases be discovered. But it is no longer doubtful that for the induction of suppurative hepatitis some form of infection must be superadded, and that the usual infective agent is the *Bacillus coli communis*. This microbe, entering the bile-channels from the bowel, sets up in them cholangitis (angiocholitis) or inflammation of the ducts, and then hepatitis as a consequence. In certain cases other parasites, such as the pyogenic micrococci, the typhoid bacillus, or the *Distomata*, gain access to the bile-ducts, and act as excitants of inflammation.

Suppurative biliary hepatitis results in the formation of **abscesses,** which at first are small and then become larger (Fig. 379 *c*), and generally contain pus that is mixed with biliary pigment or concretions. The liver sometimes is thus riddled with innumerable abscesses varying from the size of a pea to that of a cherry. If the conditions giving rise to suppuration are confined to a limited portion of the liver these biliary abscesses are circumscribed accordingly: in such cases they sometimes coalesce into one or two large abscesses, possessing characters similar to those of haematogenous origin. It is probable that many cases of tropical abscess are essentially due to biliary infection.

Biliary hepatitis is also capable of assuming a hyperplastic or proliferous character, and leads to induration of the liver or **biliary cirrhosis,** which closely resembles the haematogenous forms and is not easily distinguished from them, especially when the causes of obstruction to the outflow of bile are not at once apparent.

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CHAPTER LXXII

SYPHILIS, TUBERCULOSIS, AND LEPROSY OF THE LIVER

226. **Syphilitic disease** of the liver is common in both the inherited and the acquired forms of the affection.

Acquired syphilis may give rise to changes that are similar to those seen in ordinary cirrhosis, but it exhibits a greater tendency to induce limited and circumscribed lesions. The local lesion is inflammatory in character, and is associated with more or less extensive destruction of the liver-tissue and with fibrous hyperplasia. The characteristic result is the formation of the granulomatous nodes known as **gummata** (Figs. 380 and 381 *a*), which consist of a necrotic cheesy mass surrounded by coarse cicatricial connective tissue (Fig. 381 *b*) that sends radiating bands into the adjoining parts (*c d*). The cheesy centre of the gumma represents merely the remains of new-formed inflammatory tissue that has become necrotic and has not been absorbed. If the process at the time of examination is still incomplete, the inner and peripheral aspects of the cicatricial envelope here and there exhibit patches of granulation-tissue (*e f*), which is the forerunner of the fully-formed fibrous tissue. The necrotic hepatic tissue is in some cases entirely removed by solution

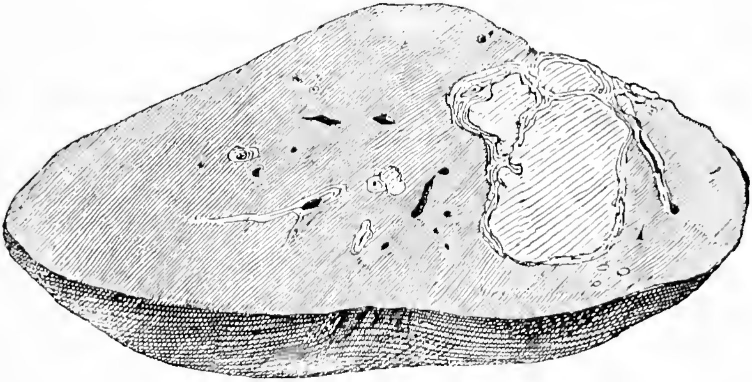

FIG. 380. SAGITTAL SECTION THROUGH THE LEFT LOBE OF THE LIVER, ENCLOSING LARGE CHEESY GUMMATA.

(*Nine-tenths of the natural size*)

and absorption, and then nothing but a shrunken fibrous scar, without any cheesy contents, remains at the site of the gumma.

Such syphilitic foci occur singly or in numbers. When the shrunken cicatrices are numerous, the liver becomes lobulated by their contraction (Fig. 372).

Congenital syphilis of the liver in certain cases produces changes resembling those of acquired syphilis, and leading to cicatricial contraction and lobulation, or to the formation of gummata. The gummata are usually found *post mortem* in an early stage of development, as circumscribed greyish patches of granulation-tissue. In some cases cicatricial fibrous bands are formed in and about the larger portal sheaths (syphilitic peri-phlebitis). But more frequently the disease leads to diffuse changes involving the entire liver, and taking the form of interlobular (Fig. 382 *a*) and intralobular (*b*) cellular infiltrations or abundant fibrous hyperplasia (Fig. 383). The cellular infiltrations do not as a rule produce any change in the naked-eye appearance of the liver, which retains its ordinary dark bluish-red

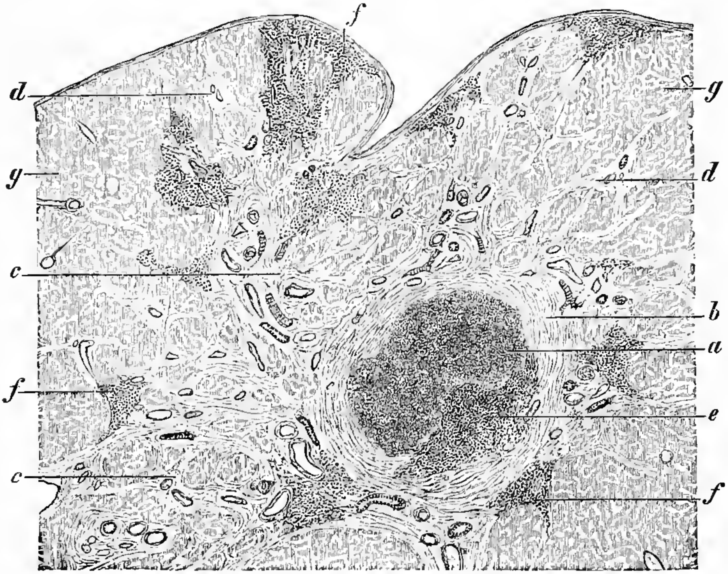

FIG. 381. SYPHILITIC CICATRIX AND GUMMA OF THE LIVER.

(*Preparation hardened in alcohol, stained with alum-carmine, and mounted in Canada balsam :* × 12)

a	cheesy centre
b	dense fibrous tissue
c	fibrous tissue enclosing remnants of liver-tissue
d	bands of fibrous tissue radiating into the liver-tissue
e	cellular (granulomatous) focus at the margin of the cheesy mass
f	cellular foci within the radiating fibrous bands
g	normal liver-tissue

colour; only when the cellular aggregations are of considerable size do they become visible as greyish nodules. It is hard to say to what extent the infiltrating cells are leucocytes brought by the blood-vessels, and to what extent they are derived from proliferous connective-tissue cells. As a certain proportion of them appear to lie within the vessels (Fig. 382 *b*), they must in part at least be regarded as white blood-corpuscles.

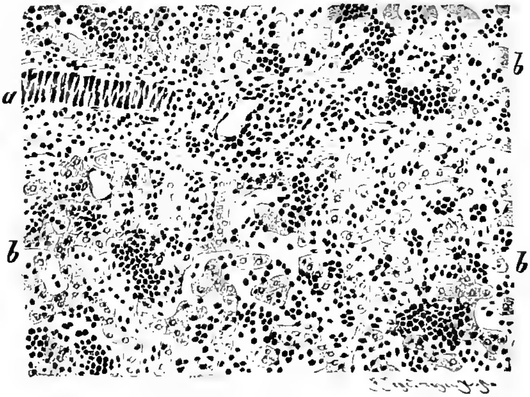

FIG. 382. SYPHILITIC HEPATITIS IN A NEW-BORN INFANT.
(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin*: × 100)

a interlobular connective tissue infiltrated with cells (longitudinal section of an artery)
b parenchymatous tissue infiltrated with cells

The second variety of the diffuse affection takes the form of fibroid induration, and in well-marked

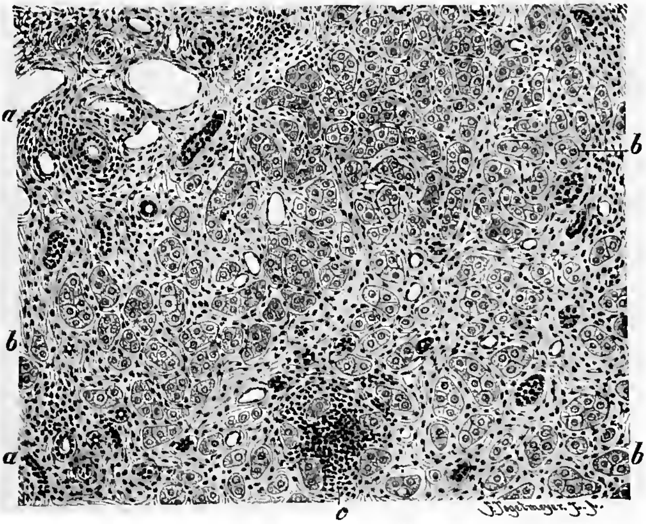

FIG. 383. INDURATED LIVER FROM A SYPHILITIC INFANT.
(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin*: × 100)

a hyperplastic interlobular connective tissue
b indurated liver-tissue traversed by fibrous bands
c clusters of round-cells

cases is easily recognisable by the pale greyish-yellow colour, resembling that of flint (GUBLER), which the liver assumes, by the indistinctness of the lobular outlines on the cut surface, and by the hardness and density of the tissue.

The fibrous hyperplasia, associated with proliferation of the biliary ducts, is most marked in the portal sheaths and interlobular tissue (Fig. 383 *a*); but it also extends to the lobules themselves, with the result that the constituent hepatic columns or trabeculae (*b*) are everywhere forced asunder and isolated by ingrowths of vascular connective tissue, and the typical structure of the lobules is thus entirely effaced.

Syphilitic disease of the liver is usually accompanied by exudative deposits upon the hepatic peritoneum (perihepatitis), and by fibrous adhesions to the adjacent structures. These are often so dense that the liver is immovably attached to the diaphragm.

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227. **Tuberculosis** of the liver is met with in three chief forms, namely as miliary tuberculosis, as tuberculous hepatitis, and as solitary tubercles.

Miliary tuberculosis is usually but a part of a general tuberculosis involving several organs or the entire body. The liver is beset with minute nodules, often so small as to be scarcely perceptible, and grey, yellow, or bile-stained as the case may be (Fig. 384). The nodules lie in the interlobular connective tissue (*a*), or within the lobules (*b*). The fatty livers of phthisical patients are sometimes thickly beset with minute tubercles that are not recognisable by the naked eye.

As the tubercles become mature the infiltrated liver-tissue in which they are seated becomes necrotic, and the cells are transformed into shapeless denucleated blocks. When a biliary duct is surrounded by or enclosed in a tubercle, its epithelial cells, especially when they are pressed together into a mass, sometimes closely simulate giant-cells. According to ARNOLD, new biliary ducts are occasionally formed in the midst of a tuberculous node. Within the larger nodes the centre usually softens and breaks down, forming small cavities that enclose puriform and often bile-stained contents.

In the second and rarer form of the affection, or chronic tuberculous hepatitis, there is not only an eruption of tubercles, but also a diffuse fibrous hyperplasia of the liver. The parenchyma is traversed by more or less dense bands of fibrous tissue, containing small grey tubercles or large yellow and often bile-stained caseous patches.

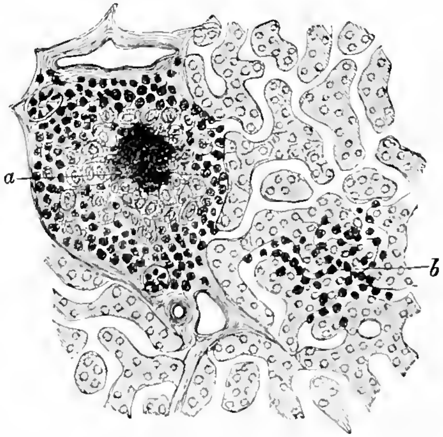

FIG. 384. MILIARY TUBERCULOSIS OF THE LIVER.

(*Preparation stained with carmine*: × 150)

a mature tubercle
b cluster of small cells forming an incipient tubercle

The third form is characterised by the development of large isolated caseous nodes, like the solitary tubercles of the brain, but the condition is very rare.

In **leprosy** foci containing the specific bacilli are sometimes observed in the liver; they lie chiefly in the interlobular portal sheaths and in the peripheral zone of the lobules. The foci consist of cellular clusters or nodes which contain the characteristic large cells crammed with bacilli.

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CHAPTER LXXIII

REGENERATION AND HYPERPLASIA OF LIVER-TISSUE

228. The liver is an organ whose tissues are capable of considerable regenerative proliferation, and this is true not only of its connective tissue, but also of its epithelial constituents.

Regenerative proliferation of the liver-cells and ducts follows not only upon degenerative changes, as we have already more than once remarked, but also upon traumatic injuries; it first makes its appearance in and about the site of the lesion.

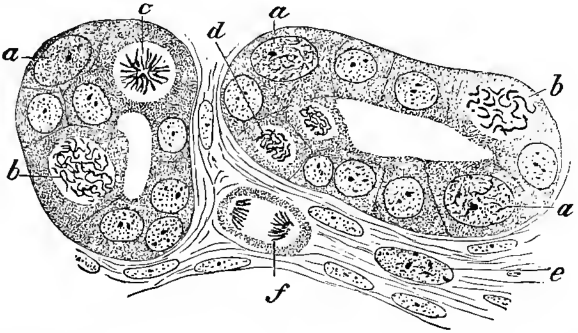

FIG. 385. REGENERATIVE PROLIFERATION OF THE EPITHELIUM OF THE BILIARY DUCTS.

(*From a wound five days old: section hardened in Flemming's acid solution, and stained with safranin and picric acid:* × 400)

a enlarged nucleus of epithelial cell with its chromatin increased
b epithelial cell with coil-stage of parent nucleus
c epithelial cell with nucleus in the star-stage (monaster)
d epithelial cell with daughter nuclei in the coil-stage
f connective-tissue cell with daughter-stars (diaster)

When the liver is injured by mechanical violence, as in the case of a punctured wound, the tissue in immediate proximity to the wound becomes the seat of haemorrhagic extravasation and inflammation; these however soon pass away, provided there is no infection capable of inducing suppuration. The reparative processes begin as early as the second day after the injury, and are indicated by the signs of mitotic nuclear division in the connective-tissue cells (Fig. 385 *f*), the biliary epithelium (Fig. 385 *a b c d*), and the liver-cells, the product of which is new-formed cellular granulation-tissue, designed to make good the loss of substance. The ultimate result of the proliferation is a cicatricial patch consisting chiefly of connective tissue and of a certain number of new biliary ducts or canaliculi, in some cases also of ill-developed liver-tissue. This patch, while it suffices to restore the continuity of the tissue, is nevertheless of little value from the point of view of functional activity.

The same phenomena are thus repeated here that are observed in the case of haematogenous and biliary degenerations, inasmuch as in the place of true secreting parenchyma nothing but non-functional fibrous tissue and abortive biliary ducts are produced (Fig. 368). Functional liver-tissue is either not reproduced at all or only in a rudimentary form.

But though such local repair of hepatic lesions is practically valueless as regards the secreting power of the new tissue, the loss, even when it is considerable, admits of complete compensation by the hyperplastic overgrowth of the undamaged parts.

The liver is a gland whose size is proportionate to the needs of the system ; in other words, its size depends on the functional calls upon it, and accordingly its bulk varies considerably in different circumstances. Whenever any considerable amount of liver-tissue is destroyed, as for example by syphilitic disease, by hydatid cysts, by haematogenous or biliary degenerations, or by surgical operation, the remaining portions of the organ, which now must carry on the whole of its work, undergo compensatory hyperplasia or **functional hypertrophy,** and so enable it to overtake the demands upon it.

According to the experimental researches of PONFICK and VON MEISTER on rabbits, even the loss of from half to three-quarters of the liver may be completely compensated by the hypertrophy of the remainder, the normal weight of the liver being almost regained in from six to eight weeks.

This compensatory hypertrophy is brought about by the enlargement of the several lobules, whose secreting cells and capillaries undergo multiplication, new elements being as it were intercalated between the old.

In man such extensive compensatory hypertrophy as is observed in the rabbit probably never takes place ; but after partial destruction of the hepatic tissue, less-marked hypertrophic enlargements are often observed, even in the course of cirrhosis and other forms of induration of the liver.

In rare cases, both in cirrhotic and in non-cirrhotic livers, the hyperplasia leads to the formation of nodose tumour-like outgrowths from the surface.

According to HOFFMANN (*Veränd. d. Organe bei Abdominaltyphus* Leipzig **1869**) there are from 110 to 116 nuclei for every 100 liver-cells. In livers in which, following degenerative changes (as in typhoid fever), regenerative multiplication is in progress, the number of nuclei increases up to from 136 to 150 for each 100 cells: for 1000 uninuclear cells there are on an average 444 containing 2 nuclei, 45 (in health only 2) containing 3 nuclei, 14 containing 4, 10 containing 5, and 1 containing 6 nuclei. The size of the cells is at the same time very unequal.

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PONFICK: Experimental researches on the pathology of the liver *V. A.* 118 **1889**, 119 **1890**, and 138 **1894**; Reproduction of the liver in man *Virchow's Festschrift* (*Assistenten*) Berlin **1891**
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CHAPTER LXXIV

TUMOURS AND PARASITES OF THE LIVER

229. **Tumours** of the liver occur both as primary and as secondary growths, the latter being frequently the result of metastasis from epithelial tumours of the intestinal tract. Among the primary growths are both epithelial and connective-tissue tumours, but the latter, with the exception of cavernous angioma, are rare.

Of the epithelial tumours **adenoma**, which may develope in normal as well as in cirrhotic livers, must first be considered; but it is on the whole a rare neoplasm. In its typical form adenoma consists of solitary or multiple greyish-white, yellowish, reddish, or brownish nodules or nodes, which are distinguished by the presence in them of tortuous branching gland-like tubules, or of new-formed trabeculae of liver-cells not grouped in the form of lobular acini. They originate either from proliferous liver-cells, or from proliferous biliary ducts. The smallest nodules are continuous with the surrounding liver-tissue; the larger ones are enclosed in a fibrous capsule, and press upon and displace the neighbouring structures.

The proliferation may be of the nature of simple hyperplasia on the one hand, and on the other may assume a malignant neoplastic character, and pass into adeno-carcinoma. It is therefore impossible to draw a sharp line between nodose hypertrophy and adenoma, or between simple adenoma and adeno-carcinoma. **Adeno-cystoma** is a special form of adenoma, whose presence is indicated by the existence in the liver of numerous cysts containing a colourless liquid, or by the transformation of entire lobes into aggregations of cysts (Fig. 386 *c d e*). Preliminary stages of these cysts may be found in the shape of more or less dilated glandular tubules derived from the biliary ducts of the interlobular connective tissue.

Carcinoma of the liver, as a primary growth, occurs in three chief forms. In the first or massive form, one (Fig. 397 *a*) or two large nodes are formed. These may be seated in any part of the liver, but appear most commonly in the right lobe. The nodes are often of great size (*a*), and consist of soft or hard white or slightly-reddened tissue: the amount of cancer-juice which can be scraped from the cut surface varies much, and is sometimes very small indeed. At some points the tumour-tissue is sharply marked off from the liver-tissue, the latter being manifestly thrust aside and the biliary ducts and vessels compressed. At other points the tumour is continuous with the liver-tissue, sometimes penetrating into the large blood-vessels or bile-ducts.

The larger nodes are frequently softened or necrotic in the centre, or contain extravasated blood. If the cancer is situated beneath the serous membrane, and a portion of the tissue in its interior is absorbed, an umbilicated depression forms on the surface of the nodule. In the course of time metastases (*b*) are developed within the liver, and the organ may thus become studded with numerous nodules (nodular cancer).

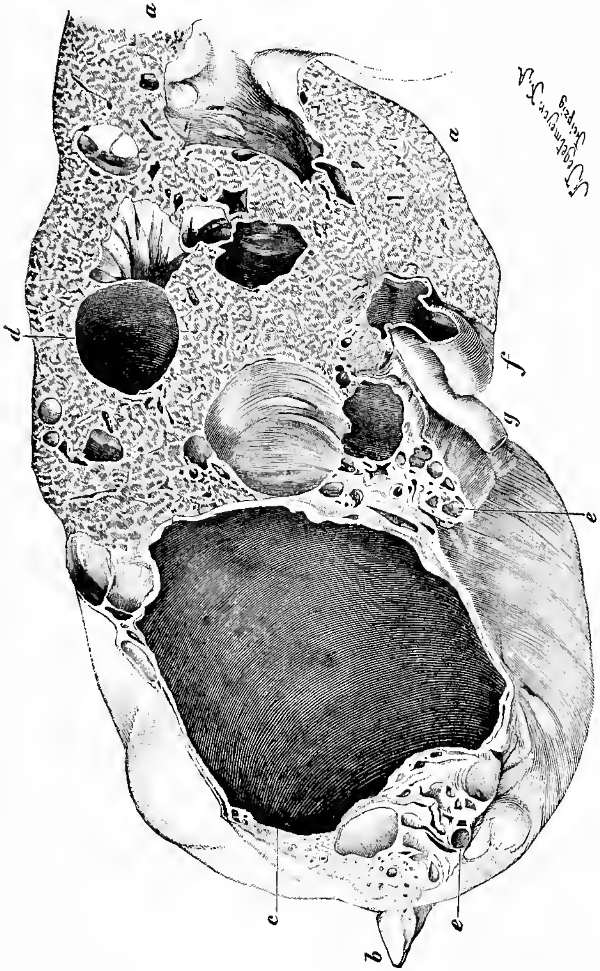

FIG. 386. ADENO-CYSTOMA OF THE LIVER.

(*Seen in section: two-thirds of the natural size*)

a	parenchyma of the liver
b	membranous border of the left lobe
c d	larger cysts
e	group of smaller cysts separated only by fibrous tissue
f	portal vein
g	hepatic artery

The second form simulates the appearance of a granular or tuberculated cirrhotic liver, islands of whitish or brownish tissue being enclosed between stout anastomosing fibrous bands that traverse the organ in all directions. These islands consist however of cancer-tissue, and the condition is sometimes described as cancerous cirrhosis or cirrhotic cancer (*cancer avec cirrhose*).

In the third form the cancerous growths are seated in the interlobular connective tissue. Wherever the portal vessels run they are seen to be accompanied by white tumid nodules, in close mutual contact or actually coalescent, and varying in size with the size of the vessels.

In structure certain varieties of hepatic carcinoma are to be classed as cylindrical-celled adeno-carcinomata; others as ordinary cancers of the simple or medullary type with solid cell-nests. They originate either from the glandular cells of the hepatic trabeculae or from the epithelial cells of the biliary ducts, the third form in particular being of the latter kind (Fig. 388). The forms associated with fibroid induration make their appearance in cirrhotic livers (HANOT, GILBERT, SIEGENBECK, VAN HEUKELOM, FROHMANN, ZIEGLER), and their mode of origin can be traced histologically by the fact that the chromatin of the liver-cells is increased, while their protoplasm is more abundant and possesses a different type of granulation and different staining-reactions from those exhibited by normal liver-cells. When the cirrhosis affects the entire liver, the adenomatous or carcinomatous proli-

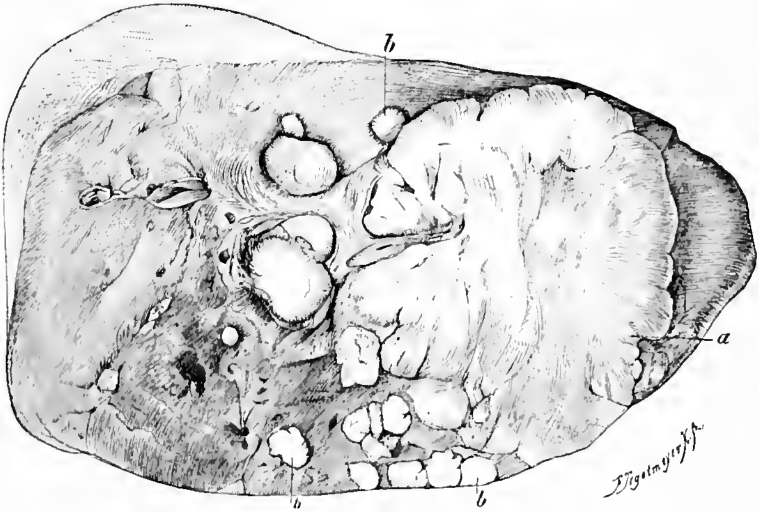

FIG. 387. PRIMARY CARCINOMA OF THE LIVER.

(*Rather less than half the natural size*)

a the primary node *b* multiple metastases within the liver itself feration likewise extends throughout its substance. If the cirrhosis is a local one, such for instance as accompanies the presence of *Distoma* in the liver, the carcinomatous growth starts first in the morbidly-altered region, but may thence spread in the form of nodular infiltrations to the healthy hepatic tissue.

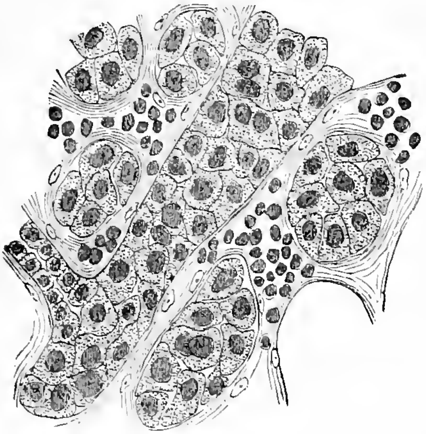

FIG. 388. CARCINOMA OF THE BILE-DUCTS.

(*Below at the left side a cancerous cell-nest communicates with a biliary duct: preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 200)

Metastatic carcinoma of the liver is most frequently secondary to carcinoma of the intestinal tract, more rarely of the uterus, pancreas, and breast. The growth starts from cancerous emboli within the vessels (Fig. 389), which as they multiply force aside the liver-tissue, or following the course of the vessels pervade the parenchyma (Fig. 390). Presently the liver-cells become atrophied and disappear, while the vascular and fibrous elements become proliferous.

The metastatic nodules may be few or exceedingly numerous; cases are occasionally met with in which the greater part of the liver is beset with miliary and sub-miliary nodules. As the disease progresses these coalesce to larger nodules and nodes having a diameter of from 0.5 to 10 centimetres.

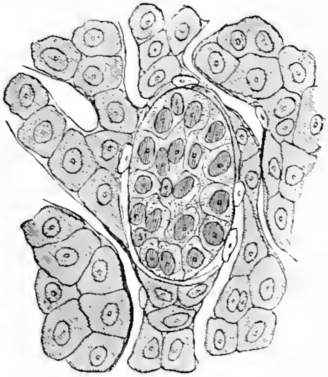

FIG. 389. CANCEROUS EMBOLUS IN A HEPATIC CAPILLARY.

(*From a primary adeno-carcinoma of the stomach: preparation stained with haematoxylin:* × 300)

The smaller growths, when they lie beneath the serous membrane, appear on the surface of the liver as small whitish patches; the larger ones project slightly above the level of the surrounding liver-surface and are frequently umbilicated. The overlying part of the serous membrane is usually much congested and injected. When the cancer-nodules are large and numerous the liver is more or less increased in size, often enormously so, and its surface is uneven and tuberous. The nodes on the anterior edge can be felt through the relaxed abdominal wall. On section the tumours appear generally white or yellowish-white, with perhaps a tinge of red.

The centre of a large node is not uncommonly found to be opaque, fatty, and softened, so that scraping yields a pulpy mass rather than a juice. The surrounding liver-tissue is visibly compressed, and it may or may not be sharply marked off from the tumour-tissue. The smaller nodes are usually ill-defined, the larger ones are more distinctly circumscribed. But in this respect something depends on the nature and structure of the primary tumour, metastases derived from adeno-carcinoma of the stomach and intestines having a tendency to thrust back the surrounding tissue, while those derived from mammary, pancreatic, and oesophageal growths infiltrate it.

The liver-tissue itself is brown, yellow, or yellowish-green, the last being a sign of retention or stagnation of bile. When the cancerous growths are numerous, the liver-tissue persists only as islands or narrow bands between the several nodes.

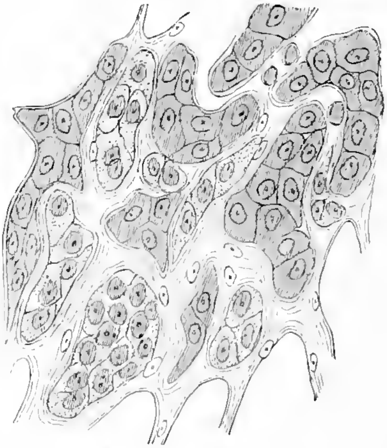

FIG. 390. METASTATIC GROWTH OF CANCER IN A HEPATIC CAPILLARY.

(From a primary carcinoma of the pancreas : stained with haematoxylin : × 250)

Heterotopic nodules of **suprarenal tissue** have in a few cases been observed in the liver.

References on Epithelial Tumours and Cystoma of the Liver (see also Art. 233 for references on Cysts).

BABES: Primary cancer *A. roumaines de méd.* I **1888**

BENEKE: Adenoma *Ziegler's Beiträge* XI **1891**

BONOME: Idem *A. per le scienze med.* XIII **1889**

BRISSAUD: Adenoma and cancer *A. gén. de méd.* II **1885**

CROOKE: Primary cancer *B. M. J.* I **1888**

DMOCHOWSKI and JANOWSKI: Total cystic degeneration *Ziegler's Beiträge* XVI **1894**

EBERTH: Adenoma *V. A.* 43 **1868**

FAGGE: Cancer simulating cirrhosis *Trans. Path. Soc.* XXVIII London **1876**

FROHMANN: Adenoma *Inaug. Diss.* Königsberg **1894** (with references)

GREENFIELD: Primary columnar epithelioma *Trans. Path. Soc.* XXV London **1874**

GRIESINGER: Adenoid of the liver *A. d. Heilk.* V **1864**

HANOT and GILBERT: *Études sur les maladies du foie* I Paris **1888** (with references)

HARRIS: Development of primary cancer of the liver *V. A.* 100 **1885**

VAN HEUKELOM: Adeno-carcinoma *Ziegler's Beiträge* XVI **1894** (with references)

von Hippel: Cystadenoma of the bile-ducts *V. A.* 123 **1891**
Hoffmann: Adenoma *V. A.* 39 **1867**
von Kahlden: Genesis of the cystic liver *Ziegler's Beiträge* xiii **1893**
Kelsch and Kiener: Adenoma *A. de physiol.* iii **1876**
Laveran: Primary cylindrical-celled epithelioma *A. de physiol.* **1880**
Martin: Carcinoma and adenoma of the liver in the horse *Jahresber. d. Thierarzneischule* Munich **1882–83**
Naunyn: Origin of hepatic cancer *Reichert's und du Bois-Reymond's Arch.* **1866**
Paul: Primary tumours *Lancet* i **1885**
Perls: Histology of cancer of the liver *V. A.* 56 **1872**
Rindfleisch: Adenoma *A. f. Heilk.* v **1864**
Rovighi: Racemose adenoma with fatal jaundice *A. per le scienze med.* vii **1883**
Schmorl: Accessory adrenals *Ziegler's Beiträge* ix **1891**
Schüppel: Carcinoma *Ziemssen's Cyclop.* ix New York **1880**
Schweizer: Cystadenoma *V. A.* 113 **1888**
Siegmund: Cystadenoma *V. A.* 115 **1889** (with full references)
Simmonds: Nodose hyperplasia and adenoma of the liver *D. A. f. klin. Med.* xxxiv **1883**
Terburgh: Cysts of the liver and kidneys *Inaug. Diss.* Freiburg **1891** (with references)
Weigert: Primary carcinoma *V. A.* 67 **1876**
White: Primary cancer *Trans. Path. Soc.* xxxvi London **1884**
Wulff: Primary cancer *Inaug. Diss.* Tübingen **1876**

230. Primary connective-tissue growths are very rarely met with in the liver; but **cavernous angioma** or erectile tumour is not infrequent. It forms nodes as small as a millet-seed, or as large as the fist, which takes the place of a corresponding amount of liver-tissue. The tumour thus produces no enlargement of the liver as a whole.

Angiomata lying close beneath the serous membrane appear as dark bluish-red spots; on section they are dark red. In the larger nodes the spongy texture is easily recognised, the lighter fibrous septa contrasting with the blood contained in the meshes and cavities. The larger nodes are bounded by a fibrous capsule, while the smaller ones are continuous with the liver-tissue.

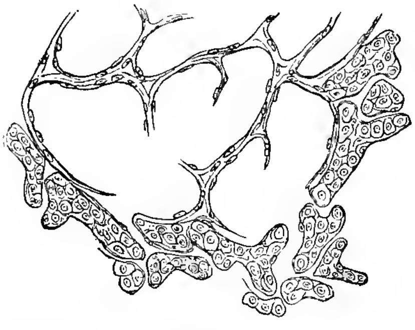

Fig. 391. Section from the growing margin of a small cavernous angioma of the liver: × 150.

Angioma of the liver occurs most frequently in the atrophied livers of aged persons, and is produced by the cavernous degeneration of the capillary network of the liver (Fig. 391), accompanied by atrophy of the liver-cells. Proliferation of the vessel-walls, of the intervascular connective tissue, and of the margins of the growth, is a secondary phenomenon. The capsule of the larger tumours is formed essentially of the interlobular connective tissue. The intercavernous septa vary in thickness, but are usually thin and delicate. Thrombosis and subsequent fibrous hyperplasia sometimes give rise to the formation of cicatricial tissue in the interior of an angioma.

Primary fibroma occurs in the form of numerous nodules and nodes developed along the course of the sympathetic nerves, and therefore belongs to the group of neurofibromata. It appears as part of a general fibromatosis extending throughout the nervous system.

Primary sarcoma of the liver is very rare, and has been described in only a few instances. The recorded observations (ARNOLD) show that it developes from the connective tissue in the hilum of the liver, from the fibrous sheaths of the larger vessels and bile-ducts, and from the interlobular connective tissue, the neoplastic growth taking place chiefly in the neighbourhood of the vessels. Nodules of varying size are formed within the liver, either singly or in large numbers, and these may give rise to sarcomatous metastases in other organs.

Secondary sarcoma takes the form either of circumscribed greyish-white nodes of large size, or of small disseminated nodules, which pass continuously into the tissue of the enlarged liver, or infiltrate it in a diffuse manner without forming any definite tumour. The microscope shows that the growths that are not sharply circumscribed cause a kind of sarcomatous infiltration of the liver-substance, the proliferous sarcoma-cells spreading along the intralobular capillaries, and as they multiply displacing the trabeculae of liver-cells that lie between them.

Metastatic melanosarcoma likewise takes the form either of circumscribed nodes or of diffuse infiltration: in the latter case it often gives rise to a peculiar mottling resembling that of granite, the tissue being sprinkled or marbled with black, yellowish-brown, and grey tints. It is still a question whether melanotic sarcoma ever occurs as a primary growth in the liver.

Cysts of the liver arising from lymphangiectasis are very rare.

References on Connective-tissue Tumours of the Liver.

ARNOLD: Primary angiosarcoma *Ziegler's Beiträge* VIII **1890**
BÉLIN: Melanosis in a case of melanotic carcinoma *France méd.* I **1887**
BENEKE: The genesis of angioma *V. A.* 119 **1890**
BERTOLET: Multiple angiomata *Trans. Path. Soc.* V Philadelphia **1876**
BLOCK: Primary melanotic endothelioma *A. d. Heilk.* XVI **1875**
BRAMWELL, BYROM: Primary sarcoma *Lancet* I **1897** (with references)
BURNET: Primary melanotic sarcoma *Trans. Path. Soc.* XXXVI London **1884**
CHERVINSKY: Multiple cavernous angiomata in an infant aged six months *A. de physiol.* **1885**
DELÉPINE: Primary melanotic sarcoma *Trans. Path. Soc.* XLII London **1890**
HANOT and GILBERT: *Études sur les maladies du foie* I Paris **1888** (with references)
LITTEN: Secondary melanotic sarcoma *D. med. Woch.* **1889**

Moore: Melanotic sarcoma *B. M. J.* I **1889**
Orth: Primary sarcoma *Inaug. Diss.* Strassburg **1885**
Pisenti: Multiple fibromata *Lavori compiuti nell' Istituto anatomo-patologico* Perugia **1886**
Podrouzek: Cases of sarcoma *Prag. med. Woch.* **1888**
Tooth: Lymphosarcoma *Trans. Path. Soc.* XXXVI London **1884**
Windrath: Sarcomatosis (case of primary spindle-celled sarcoma) *Inaug. Diss.* Freiburg **1885**

231. Among the **animal parasites** that take up their permanent abode in the liver are the following: *Echinococcus* (hydatid), *Distoma hepaticum*, *Distoma lanceolatum*, *Distoma haematobium*, *Pentastoma denticulatum*, and the so-called *Psorospermia*. These parasites are described in the volume on General Pathological Anatomy: here we propose merely to add something concerning the *Echinococcus* or hydatid of the liver.

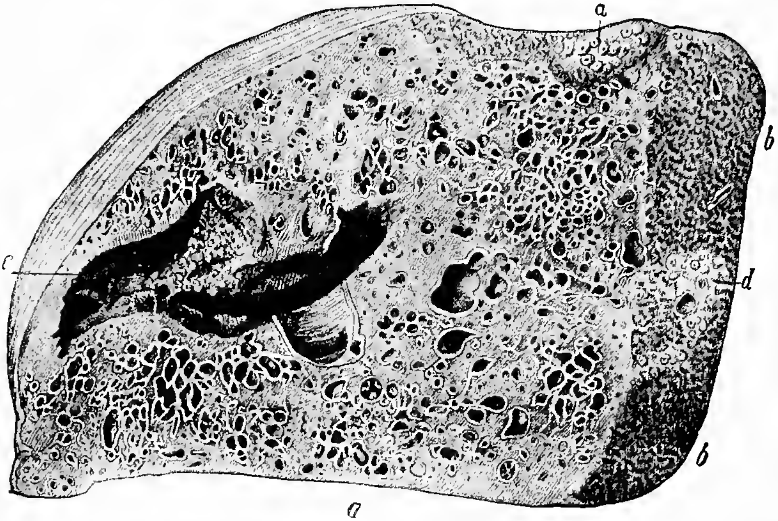

Fig. 392. Echinococcus multilocularis in section.
(*Natural size*)

a alveolar structure produced by the *Echinococcus*
b liver-tissue
c cavity due to softening
d recent nodules

The **hydatid** has usually the form of a simple chitinous cyst of the size of a walnut or larger, which is surrounded by a fibrous capsule. Cases in which the mother-cyst is accompanied by exogenous or endogenous daughter-cysts are less common.

By the time the liver comes to be examined the *Echinococcus* is usually dead, and the intracystic liquid wholly or partially absorbed. In this case the chitinous cyst is shrunken and folded, and contains a white cheesy, pulpy, or mortar-like and calcareous mass, within which the characteristic hooklets may often be discovered. When the cysts reach a large size they may burst into the peritoneum or intestine, or externally. Sometimes inflammation is set up around the cysts, or retention of bile and biliary hepatitis are induced.

Echinococcus multilocularis (Fig. 392) gives rise to hard nodes occupying the greater portion of a lobe, and sometimes causing considerable enlargement of the affected part. They consist of dense tough whitish or somewhat bile-stained fibrous tissue, enclosing numerous alveoli of various sizes filled with jelly-like matter. When the tumour exceeds the size of a man's fist, the central portions usually undergo softening. As the *Echinococcus* grows, yellowish or pale yellowish-brown specks (*d*) appear in its neighbourhood, which soon become converted into small cysts with firm walls and colloid contents, and coalesce with the neighbouring cysts. When the central portions soften and break down, cavities of very considerable size are sometimes produced (*c*).

CHAPTER LXXV

THE GALL-BLADDER AND BILE-DUCTS

232. **Biliary concretions** and **gall-stones** are by far the commonest abnormalities in the contents of the gall-bladder and bile-ducts (hepatic, cystic, and common). They occur very frequently in elderly persons, more particularly within the gall-bladder. The concretions are friable granular yellowish-brown or black masses. The gall-stones or biliary calculi, which may be as small as a millet-seed or as large as a hen's egg, are in some cases rounded or ovoid, in others angular and facetted (Fig. 393). The latter condition is found when several calculi have lain together in contact within the gall-bladder or the bile-ducts; the former when they have lain singly.

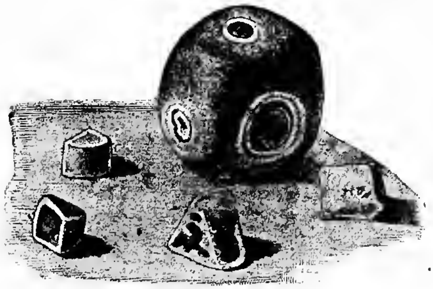

FIG. 393. FACETTED GALL-STONES. (*Natural size*)

The colour, consistence, and density of the stones vary with their composition. As a rule they are somewhat soft, the surface pale greyish-white, yellowish deepening to dark-brown, or mottled, and either smooth or rough as the case may be.

On section there is usually a dark-coloured nucleus consisting of calcareous pigment (calcic bilirubin), surrounded by a lighter shell or crust containing crystalline plates of cholesterin radially disposed. The different species are distinguished according to the constituent which is most abundant.

(1) Stones consisting of **cholesterin** contain a pigmented nucleus, are single or multiple, greyish-white or yellowish in colour, smooth or rough on the surface, slightly translucent, with sometimes a kind of pearly lustre. The fractured surface has a radiate crystalline appearance, and often shows traces of stratification. The stones are soft in texture. When stained with bile they are of a yellowish or brownish colour. When mixed with calcium-salts they are friable and chalky.

(2) Stones consisting of **cholesterin and bile-pigment** are

the commonest of all. According to their proportion of pigment they are yellow, brown, black, or brownish-green: at times the successive strata are of different tints. They are frequently present in enormous numbers, and distend the gall-bladder and ducts to a remarkable extent. They sometimes contain a considerable amount of calcium carbonate and of magnesium-salts.

(3) Pure calcic **bilirubin** and **biliverdin** stones are rare and usually small; they have a rusty-red or almost black colour.

(4) Stones consisting of **calcium carbonate** are very rare.

The formation of gall-stones is due to incrustation upon an organic substratum or nucleus, the material for which is furnished by the mucous membrane of the bile-ducts and the gall-bladder. Accordingly we find that after the cholesterin and the calcareous pigment have been dissolved out of a gall-stone, a nitrogenous body remains in the form of a stratified stroma (Fig. 394), in whose meshes and clefts the crystalline material had been deposited.

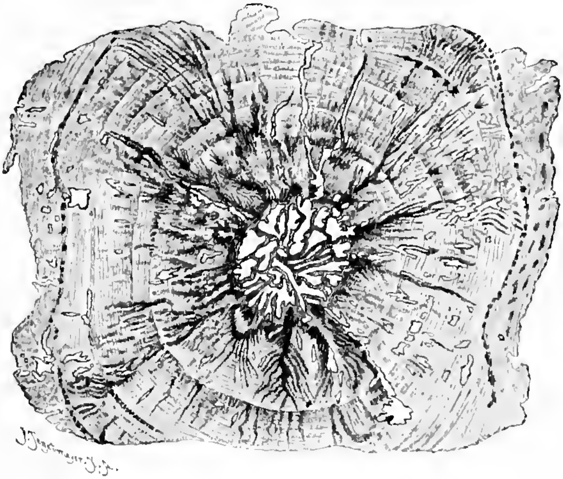

FIG. 394. SECTION THROUGH A SMALL FACETTED STONE AFTER REMOVAL OF THE CHOLESTERIN: × 15.

Gall-stones are much more common in patients of advanced age than in younger patients. Stagnation and decomposition of the bile, and inflammation of the ducts and gall-bladder, favour their production. The material for their formation is derived chiefly from desquamated and disintegrated epithelial cells.

The effects of gall-stones (**cholelithiasis**) vary greatly in different cases. Often the gall-bladder is affected very slightly, even when it contains a large number of stones. In other instances the wall of the gall-bladder is the seat of intense inflammation, resulting in ulceration or induration, sometimes even in suppuration and abscess, when secondary infection by the *Bacillus coli communis* or pyogenic micrococci takes place. At a later stage, hepatic abscess, perforation, purulent peritonitis, or the like, may supervene.

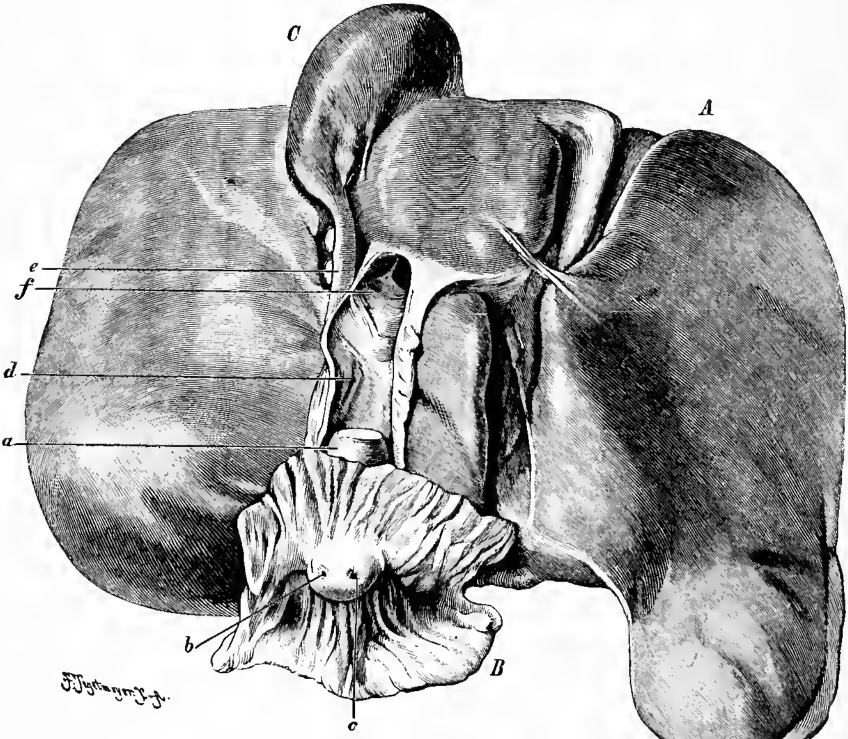

FIG. 395. OCCLUSION OF THE COMMON DUCT BY GALL-STONES AND DILATATION OF THE HEPATIC DUCT.

(*Under surface of the liver with a bit of the duodenum laid open: one-half the natural size*)

A	liver
B	portion of the duodenum
C	gall-bladder
a	gall-stone
b	mouth of the common duct pushed forward into the lumen of the bowel by the gall-stone
c	attenuated and almost perforated wall of the bowel
d	dilated common duct
e	cystic duct
f	dilated hepatic duct

Stones formed in the gall-bladder and in the ducts themselves are frequently discharged into the intestine through the common duct. But if one of these lodges or lingers in the duct (Fig. 395 *a*), retention of the bile ensues, and this gives rise to dilatation of the ducts (*d*, *f*) and icteric pigmentation of the liver. As a later result degeneration of the liver-cells and biliary hepatitis are apt to be set up (Art. 225). When inflammation and ulceration are produced in the neighbourhood of a calculus the duct may become perforated, and intense inflammation is then excited in the surrounding tissues. If the stone is impacted near the mouth of the common duct in the duodenum, the ulceration of the duct and of the bowel-wall (Fig. 395 *c*) may set it free, and permit it to pass into the intestine. A stone may also escape directly from the gall-bladder into the duodenum or colon when previous inflammatory adhesions have been formed between the bladder and the bowel, and ulceration opens through these an abnormal communication.

When concretions form in the ducts within the liver, more or less intense inflammatory change is frequently set up around them. The slighter forms give rise to moderate cellular infiltration of the duct-wall and adjoining tissue, and this after a time results in the formation of new fibrous tissue. In other cases, when infection is superadded, the inflammation becomes suppurative and ends in an abscess (Art. 225).

References on Gall-stones and their Effects.

FRERICHS: *Diseases of the liver* II London **1862**
JANOWSKI: Changes in the gall-bladder induced by gall-stones *Ziegler's Beiträge* X **1891**
KONITZKY: Cystic dilatation of the ductus choledochus *Inaug. Diss.* Marburg **1888**
MARCHAND: On a common cause of gall-stones in women *D. med. Woch.* **1888**
MAYER: Experimental researches on the formation of gall-stones *V. A.* 136 **1894**
NAUNYN: Diseases due to gall-stones *Verh. Congress. f. inn. Med.* Wiesbaden **1891**; *Klinik der Cholelithiasis* Leipzig **1892** (trans. by GARROD) London **1896**
ORTNER: *Klinik d. Cholelithiasis u. d. Gallenwegsinfection* Vienna **1891**
PETERSSEN-BORSTEL: The relation of gall-stones to cancer and endarteritis *Inaug. Diss.* Kiel **1883**
POSNER: Studies on the structure of calculi *Z. f. klin. Med.* IX, and *D. med. Woch.* **1880** and **1886**
RIEDEL: *Erfahrungen über Gallensteinkrankheit* Berlin **1892**
ROTH: Diseases due to gall-stones *Correspbl. f. Schweizer Aerzte* XI **1881**; Cholelithiasis *Virchow's Festschrift* (*Assistenten*) Berlin **1891**
SCHÜPPEL: *Ziemssen's Cyclop.* IX (with references)

233. **Inflammation** of the bile-ducts and the gall-bladder, apart from that due to calculous lesions, may be of haematogenous origin or conducted from contiguous parts. The exciting agent in the latter case may penetrate into the ducts or the bladder from the bowel, the liver itself, the peritoneum, or the viscera about the porta of the liver. Among the bacteria the *Bacillus coli communis*, the pyogenic micrococci, the typhoid-bacillus, the *Diplococcus pneumoniae* and the tubercle-bacillus have been demonstrated in the bile-ducts and the gall-bladder. Of the animal

parasites liver-flukes (*Distoma*) and round-worms (*Ascaris*) have been found in the hepatic ducts.

The inflammation generally presents the character of mucous or muco-purulent catarrh. Severer forms may lead to suppuration or to diphtheritic or gangrenous necrosis of the mucous membrane and the adjacent structures.

The results of the inflammation include, in different cases, contraction with induration and perforation of the gall-bladder and ducts, together with adhesions and suppuration of the surrounding parts (hepatic and pancreatic abscess) and general peritonitis.

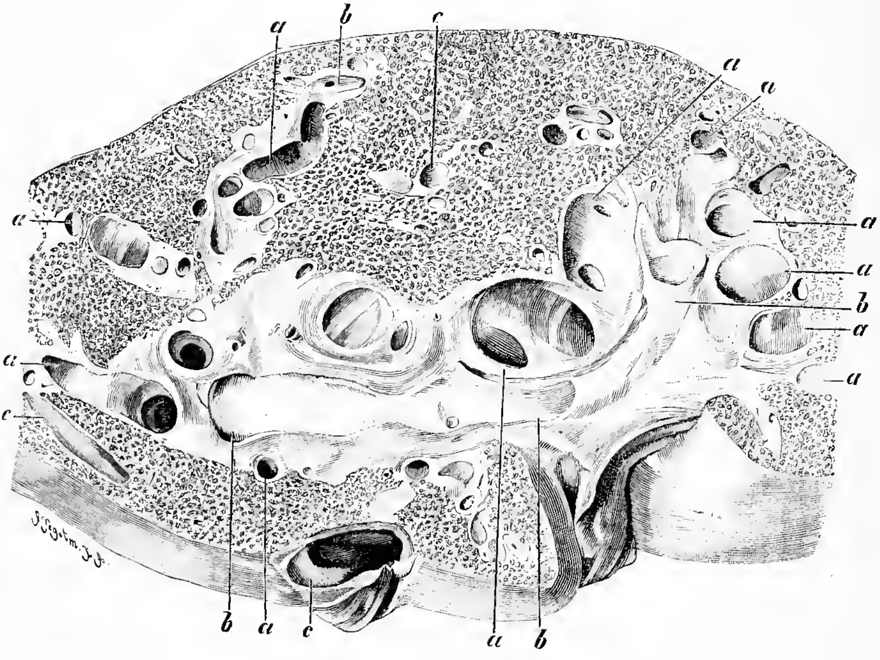

FIG. 396. DILATATION OF THE BILE-DUCTS.

(*Due to extreme constriction of the ductus choledochus by a secondary cancerous growth in the duodeno-hepatic ligament (the primary cancer being in the gall-bladder): vertical section through the liver: eleven-fourteenths of the natural size*)

a dilated ducts *b* portal vein *c* hepatic vein

Constriction and **occlusion** of the bile-ducts are produced by swelling of the mucous membrane, accumulation of secretion, gall-stones or round-worms in the lumen, cicatricial contraction of the duct-wall following ulceration, neoplastic growths in the duct-wall, and compression from without due to neoplasms, abscesses, aneurysms, enlarged lymphatic glands, or induration of the surrounding tissues. When the ductus choledochus or the hepatic duct is obstructed retention of bile and biliary engorgement are the result.

Dilatation of the ducts is generally due to constriction at a lower point, and is effected by the accumulation of the bile and the secretion of the mucous membrane. If the obstruction is in the ductus choledochus, the larger and middle-sized intrahepatic ducts sometimes become so dilated that the liver seems tunnelled with wide channels, exceeding the portal vein in calibre (Fig. 396 *a*).

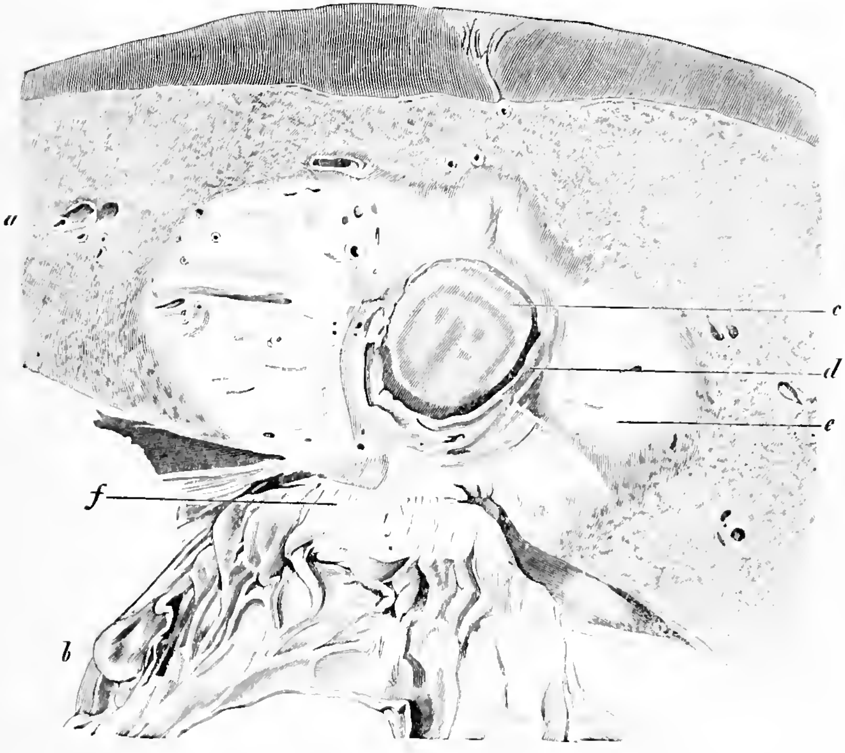

FIG. 397. PRIMARY CANCER OF THE GALL-BLADDER ENCLOSING A GALL-STONE.

(*Vertical section through gall-bladder and liver: natural size*)

a liver
b duodenum
c gall-stone
d wall of the carcinomatous gall-bladder
e carcinomatous infiltration of the adjacent liver-tissue
f portion of duodenum infiltrated with carcinoma and adherent to the primary growth

In addition to such wide-spread dilatations of the ducts, we also meet with circumscribed cystic excavations in the liver, the **cysts** being thin-walled, and varying from the size of a pin-head to that of a fist, or even larger. These are either buried within the liver-substance or project above the surface; sometimes they even hang pendulous like sacs within the abdominal cavity. As the contents of these cysts form a clear liquid containing no bile, the condition is due not to dilatation of ordinary functional ducts, but to enlargement of aberrant non-functional tubules. In some cases, particularly when the cysts are multiple, the condition is one of adeno-cystoma (Fig. 386). The walls of the cyst are lined with ciliated cylindrical epithelium or with flat pavement-epithelium.

When the cystic duct is occluded, so that no bile enters the gall-bladder, the bile it contains is absorbed, and the gall-bladder contracts. Often, however, the contrary result ensues, and the bladder, after the bile is absorbed, becomes distended by the accumulation in it of clear or slightly-turbid mucous secretion, a condition termed **dropsy of the gall-bladder** or *hydrops vesicae felleae*.

Of the **tumours** of the gall-bladder and the larger bile-ducts, the most important is **carcinoma**, whose appearance in nearly every case is referable to the presence of calculi (Fig. 397 *c*).

The growth, which is of the cylindrical epitheliomatous type, takes the form of a papillary or fungous tumour, or of a cancerous ulcer from whose floor start thickenings and indurations of the bladder-wall due to neoplastic infiltration (*d*). From the gall-bladder the cancer may invade the liver (*e*), the duodeno-hepatic ligament, and finally the duodenum. In districts where gall-stones are common, hepatic cancer is not infrequent.

It has already been mentioned (Art. 239) that carcinoma may originate in the bile-ducts.

The other tumours found in the gall-bladder and ducts include sarcoma, myxoma, and fibroma, but they are all very rare.

References on Affections of the Bile-ducts and Gall-bladder (see also Arts. 229 and 225)

Bard and Lemoine: Essential cysts of glandular organs *A. gén. de méd.* **1890**
Charcot: Primary cancer of bile-ducts *Progrès méd.* IV **1876**
Combis: *Contrib. à l'étude des kystes du foie et des reins* Paris **1877**
Courvoisier: *Beiträge z. Pathologie u. Chir. d. Gallenwege* Leipzig **1890**
Eberth: Cysts with ciliated epithelial cells *V. A.* 35 **1866**
Friedreich: Idem *V. A.* 11 **1856**
Hanot and Gilbert: *Étude sur les maladies du foie* I Paris **1888**
Heddaeus: Tumours of the gall-bladder *Beiträge von Bruns* XII **1894** (with references)
Juhel-Rénoy: Cystic degeneration of the liver and kidneys *Rev. de méd.* I **1881**
Lomer: Congenital obliteration of the bile-ducts *V. A.* 99 **1885**
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CHAPTER LXXVI

THE PANCREAS

234. The **pancreas** is a compound racemose gland situated within the abdominal cavity, whose duct (the canal of Wirsung) passes into the wall of the duodenum and there joins the common bile-duct, opening with it through a common orifice into the bowel. Only in rare cases has it a distinct orifice. The secretion of the pancreas, which is in effect an abdominal salivary gland, acts powerfully in promoting digestion not only of starchy matters but also of albuminoids and of fat.

Of developmental **anomalies** the presence of an accessory pancreas is the most noteworthy. This takes the form of a discoid or rarely a cylindrical mass, from the size of a lentil to that of a silver crown-piece or dollar, made up of glandular lobules, and usually seated in the wall of the upper, more rarely in that of the lower, part of the duodenum or of the stomach (NAUWERCK). It is generally concealed in the wall of the bowel, but may project beyond the surface. Its minute structure is exactly similar to that of the pancreas itself, and it communicates with the intestinal canal by a duct of its own. The division of the pancreas into two equal or unequal lobes is much less common. Absence of the pancreas has been observed in ill-developed foetuses, in which either the entire body or the intestinal tract alone was gravely malformed.

Haemorrhage (pancreatic apoplexy) within the pancreas is not very infrequent as the result of venous engorgement, of atheromatous change in the arteries, of necrosis, of crushing, laceration, and other traumatic injuries of the gland, or of haemophilia. It at times attains a considerable magnitude, both the intra-pancreatic connective tissue and the adipose tissue becoming soaked with blood, and haematomata or blood-cysts being produced in the substance of the gland. Extensive pancreatic haemorrhage may prove fatal, probably through its effect on the solar plexus. The absorption of the extravasated blood is associated with induration or cystic excavation of the damaged parts.

Atrophy of the pancreas is met with chiefly as a senile change, but it has been observed in younger patients, especially in such as have suffered from diabetes.

Fatty degeneration of the glandular cells is likewise met with, and is characterised by the pale cloudy or yellowish-white tint it imparts to the parenchyma. It occurs in the course of certain infective and toxaemic conditions.

Amyloid degeneration of the connective tissue of the pancreatic blood-vessels makes its appearance under the same conditions as in the liver, spleen, and kidney.

A peculiar form of **necrosis** associated with the deposition of calcareous salts not infrequently affects the adipose tissue of the pancreas, and may thence extend to the glandular substance. It is indicated by the appearance of dull white patches in the fatty tissue, and in rare cases is widely diffused. Softening and excavation of the tissue follows, and haemorrhage into the cavities so produced is not uncommon.

The cause of the necrosis lies probably in malnutrition of the pancreatic tissue, due to interference with its circulation, as for instance in cases of arterial sclerosis or excessive adiposity. It is possible, however, that in some of the cases infection and inflammation have had a share in the causation.

Histologically, the necrosis is characterised by the disappearance of the nuclei of the fat-cells and ultimately of the glandular cells also, and by a tendency of the tissue generally to stain blue with haematoxylin. The fat of the fat-cells is rapidly disintegrated, its liquid portions being removed, while the solid fatty acids remain and combine with calcium to form oleates. In the parts adjoining the necrosis proliferous inflammation is usually set up, and this may result in the replacement, encapsulation, or exfoliation of the necrotic tissue.

Inflammation of the pancreas (pancreatitis) arises most frequently from the invasion of the organ by way of the ducts by bacteria (*Bacillus coli communis*, pyogenic micrococci, typhoid-bacilli), or from extension of inflammation from the contiguous structures, in particular from the stomach (as in gastric ulcer) and the bile-ducts. Haematogenous inflammation is however also possible.

Purulent and gangrenous inflammations lead to the formation of **abscesses**, with more or less extensive destruction of the glandular tissue: cases are indeed recorded in which the entire pancreas has thus become necrotic and sloughy. Slighter degrees of inflammation recover without leaving any permanent change, though they sometimes lead to partial atrophy and induration of the organ.

Chronic inflammations producing induration or **cirrhosis** of the organ are most commonly due to extension of inflammatory processes from the stomach, and especially from gastric ulcers: very often indeed the base of a perforating gastric ulcer is formed by the inflamed and indurated pancreas.

Another cause of chronic inflammation is the formation of **concretions** and calculi, which may be single or multiple, and at times are present in large numbers within the ducts (Fig. 398 *e g*). The calculi are either smooth and ovoid or branching and tuberculated. Small concretions are sometimes diffused in the form of sandy grains throughout the glandular substance.

The formation of pancreatic calculi and concretions is due to inflammation of the ducts, which in its turn gives rise to disturbances of secretion and to fresh inflammation, resulting ultimately in partial or total destruction of the glandular elements combined with fibroid induration (Fig. 398 *b c f*). When infection supervenes upon this condition, suppuration is likely to be induced.

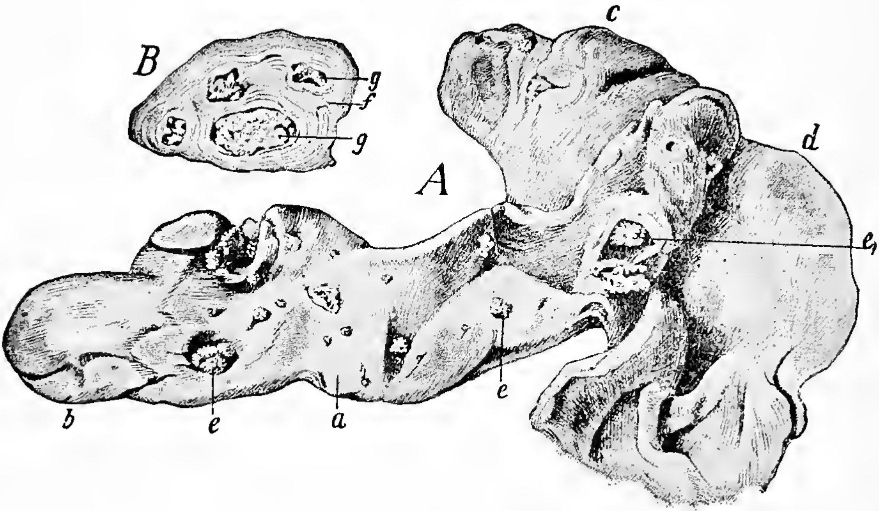

FIG. 398. CIRRHOTIC PANCREAS WITH CONCRETIONS.

(*Four-fifths of the natural size*)

A	pancreas seen from above, the main duct being laid open
a	the dilated duct studded with concretions (*e*) lodged as a rule at the openings of the secondary ducts
b	tail of the pancreas
c	lateral lobe
d	duodenum cut open
$e\ e_1$	concretions
B	transverse section of the lateral lobe *c*
f	fibroid (cirrhotic) stroma
g	dilated ducts filled with concretions

When the pancreatic duct or one of its branches is occluded by cicatrices, by compression from without, or by accumulation of secretion within, it is dilated into moniliform or sacculate cysts which are termed pancreatic **ranulae** (Fig. 399 *b*).

The position of the cysts depends upon that of the dilated duct. They are commonest in the head of the pancreas, where they are dilatations of the duct of Wirsung. Cysts of the lateral branches are rarer. The contents of a ranula are usually clear, but sometimes they are puriform, or inspissated and calcareous.

Cysts of disintegration or softening are occasionally formed at the site of necroses and haemorrhages.

Syphilis and **tuberculosis** seldom give rise to affections of the pancreas, though the specific lesions characteristic of the respective diseases are occasionally met with in the organ.

Among primary tumours of the pancreas **carcinoma** is the most frequent, and usually presents itself in the form of hard dense nodes, situated in the head of the gland. Soft medullary or colloid cancers are rare, as are all forms affecting the middle part or the tail. Sometimes, however, a carcinoma starting in the head invades the whole gland, and converts it into a single tumour that is occasionally of considerable size. The carcinomatous infiltration may also extend into the surrounding tissues, invading thus the common bile-duct, duodenum, stomach, gall-bladder, spinal column, adjacent lymph-glands, peritoneum, liver, omentum, etc. When the common bile-duct is attacked, retention of bile and jaundice are the usual result; and the closure of the duct of Wirsung in the head of the organ may produce cystic dilatation of the ducts in the tail. The adjacent veins, such as the inferior vena cava, portal vein, and superior mesenteric vein, are sometimes encircled and gripped by the cancerous growth, the result being thrombosis or other serious disturbance of the circulation.

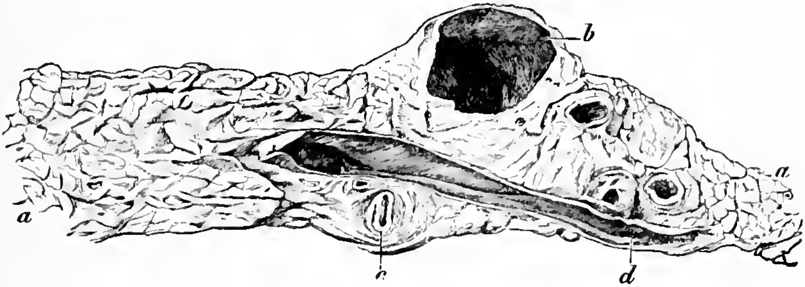

FIG. 399. PANCREATIC RANULA.
(*Longitudinal section: natural size*)

a pancreas
b cyst
c superior mesenteric artery
d splenic vein

Primary sarcoma of the pancreas is extremely rare.

Of secondary growths carcinoma is again the most important. Most frequently it is simply due to extension from cancer of the stomach or the duodenum. Metastases derived from cancer in more remote organs are much less common.

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SECTION XI

THE RESPIRATORY SYSTEM

CHAPTER LXXVII

THE NOSE AND ITS ACCESSORY CAVITIES

235. Congenital **malformations** of the nose that are at all extreme are practically met with only in combination with other deformities of the face. Thus in *Cyclopia* the nose may be absent or represented by a snout-like projection above the single orbit. Among the minor anomalies are absence of some of the turbinate bones, defects of the septum, of the ethmoid, or of the nasal bones, constriction or occlusion of the posterior nares, distortion and flexure of the nasal septum, and clefts of the alae nasi or of the floor of the nostrils.

Haemorrhage from the nasal mucous membrane (**epistaxis**) is very common, and may be due either to diapedesis or to rupture of blood-vessels. In many persons epistaxis is habitual; otherwise it occurs most frequently in connexion with the haemorrhagic diathesis, and in various infective diseases, menstrual disorders, local venous engorgements, inflammatory conditions, etc.

Inflammation of the nasal mucous membrane (**rhinitis**) is one of the commonest affections. It usually takes the form of a mucous or purulent catarrh, the croupous, diphtheritic, phlegmonous, and ulcerative varieties being much less common. The causes of mucous catarrh are largely unknown. In the severer forms of inflammation, the ordinary pyogenic bacteria are frequently found to be present, and many inflammations of the nose are associated with specific infections, such as measles, diphtheria, influenza, scarlatina, etc.

Acute nasal catarrh is spoken of as **coryza**, and is apt to result from a great variety of causes.

Chronic nasal catarrh occurs chiefly in persons who are scrofulous, phthisical, or syphilitic; it is comparatively rare in persons otherwise healthy. Sometimes it results in thickening, sometimes in attenuation and atrophy, of the mucous membrane. In the latter case the nasal cavities appear abnormally large, and their mucous membrane discharges a purulent yellowish or greenish secretion, which in the presence of certain micro-organisms undergoes decomposition and diffuses a peculiar foetid odour (**ozaena simplex**), with the formation of dirty greenish and mottled scales and crusts. According to SCHUCHARDT, in ozaena the ciliated epithelial cells of the mucous membrane disappear, and are replaced by stratified squamous epithelium. E. Fränkel points out that in the atrophic membrane the glands of Bowman are generally destroyed. In many chronic cases the bone underlying the mucous membrane likewise undergoes atrophic changes. Fränkel thus defines simple ozaena as a chronic atrophic foetid rhinitis.

Croupous and **diphtheritic inflammations** are usually secondary to like affections in the pharynx, but they sometimes begin in the nose itself.

Phlegmonous inflammation with suppuration of the mucous membrane is generally associated with inflammation of a similar character in the neighbouring parts, though it is occasionally confined entirely to the nose.

The initial sclerosis of **syphilis** appears but rarely in the nose: syphilitic catarrh (*coryza syphilitica*) is somewhat more common, and is accompanied by the formation of erythematous spots and papules which at times break down and ulcerate, causing necrotic exfoliation of the underlying portions of cartilage and bone. Gummatous inflammation is still more frequent, and starts either in the nasal mucous membrane, or in the periosteum or perichondrium of the bone and cartilage. It usually leads not only to deep ulceration of the soft parts, but also to more or less extensive destruction of the nasal bones and cartilages and of the adjacent bones of the face. The nose thus becomes depressed and distorted in various ways, and as the ulcers cicatrise and contract it sometimes collapses entirely.

The diseased mucous membrane, when the gummatous infiltration is of the diffuse kind, discharges a horribly offensive puriform secretion, which dries into a dirty-looking crust. The condition is accordingly described as **syphilitic ozaena.**

Tuberculosis of the nose is manifested by the production of the characteristic granulomatous infiltration and ulceration, which occasionally lead to caries of the bones and to a foetid puriform discharge (**tuberculous ozaena**). Lupus of the face is liable to extend to the nasal mucous membrane, and there leads to infiltrations that subsequently undergo ulceration.

Nasal **glanders** gives rise to purulent or sanguinolent catarrh, with the formation of circumscribed nodes or diffuse infiltrations in the mucous membrane. These suppurate and break down into a number of small erosions that presently coalesce into large sinuous or serpiginous ulcers. The ulcers often extend downwards to the bone, which is thereby exposed and becomes necrotic.

In **leprosy** nodes make their appearance in the nose, and subsequently ulcerate. In **rhinoscleroma** tuberous and nodular growths are produced (Art. 157).

All the inflammatory affections of the nose may extend by continuity to its accessory cavities, and there follow a more or less independent course. The cavities thus attacked become distended with mucous or purulent secretion. In severe forms of inflammation or in chronic infections (as in tuberculosis) the neighbouring bones are liable to become similarly affected. From the frontal and ethmoidal sinuses the inflammation may extend into the interior of the cranium, and so give rise to meningitis.

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236. The mucous membrane of the nose and its accessory sinuses is not infrequently the seat of hyperplastic growths and tumours, due partly to chronic inflammation, partly to undetermined causes. They take the form of diffuse thickenings or of polypous excrescences, and are usually referred to as **nasal polypi.**

Soft or mucous polypi resemble the mucous membrane in structure, but are somewhat more cellular. Sometimes the mucous glands enclosed in the growing tissue are dilated into cysts (cystic polypi), especially in the case of those appearing in the antrum of Highmore: in other cases the glands are hypertrophied and multiplied (adenomatous polypi), or the blood-vessels are abundantly developed (telangiectatic polypi or **erectile tumours**). Others again consist of oedematous connective and mucoid tissue, and are therefore classed as fibromata and myxomata. They are more translucent than the soft polypi, and are usually of a yellowish tint, while the latter are grey or greyish-red.

Sarcoma, hard fibroma, osteofibroma, chondroma, osteoma, carcinoma, and dermoids, as well as mixed tumours of the connective-tissue group, have all been met with in the nasal cavities. Many of these grow not from the mucous membrane but from the periosteum or the bone.

Connective-tissue tumours, especially those which originate in the periosteum, sometimes reach a considerable size, distending the cavity in which they grow, and sometimes protruding from its orifices.

Carcinoma of the nose is most frequently met with about the anterior nares, and is therefore classed with the cutaneous forms of cancer. Cancers originating in the mucous membrane take the form of irregularly-nodulated growths, which sooner or later ulcerate.

Rhinoliths are concretions usually of a calcareous nature encrusted round some foreign body which has become impacted in the nasal cavity. More rarely they are due to inspissation of the nasal secretions.

Of the **vegetable parasites** various forms are found in the nose, such as bacteria, moulds (*Aspergillus*), and the thrush-fungus. According to von Besser and others, the healthy nasal cavities not infrequently harbour certain pathogenic bacteria, especially *Diplococcus pneumoniae*, *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*, and *Bacillus pneumoniae*. It must moreover be borne in mind that many of the inflammations of the nasal passages are caused by specific pathogenic bacteria.

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CHAPTER LXXVIII

THE LARYNX

237. Entire absence of the larynx is a very rare **malformation.** It is met with only in amorphous and acephalous acardiac monsters. Congenital defects, as of the epiglottis or of part or the whole of the laryngeal cartilages, are much commoner. Asymmetry and abnormal largeness or smallness of the larynx are also met with. Abnormal smallness frequently accompanies aplasia of the testes, or early castration. Sometimes there are supernumerary laryngeal cartilages, and the epiglottis may be more or less deeply cleft. Not infrequently the sinuses of Morgagni are abnormally dilated, and occasionally there are extra-laryngeal pouches communicating with them.

Of acquired **abnormalities,** laryngeal stenosis is the most noteworthy. It may be caused by pressure from without, but is more commonly due either to disease of the larynx itself, such as inflammation, whereby the mucous membrane becomes swollen and covered with a membranous exudation or undergoes cicatricial contraction, or to the growth of intra-laryngeal tumours. Functional stenosis may be brought about by paralysis of the muscles which open the glottis, or spastic contraction of those which close it. Foreign bodies impacted in the glottis may have the like effect.

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238. **Laryngeal catarrh** is characterised by redness and swelling of the mucous membrane, together with a mucous, puru-

lent, or serous exudation; the latter is observed chiefly in catarrhs due to persistent venous engorgement. The inflammation may extend over the entire organ, or be limited to certain parts, such as the vocal cords or the epiglottis, and may be induced by the most diverse causes. The inflamed tissue undergoes more or less abundant cellular infiltration.

Destruction of the epithelium and suppuration of the connective tissue sometimes give rise to erosions and ulcers. These appear oftenest in and about the vocal cords and in the posterior commissure, and are due for the most part to the settlement of bacteria in the loosened epithelium.

In chronic catarrh the blood-vessels become permanently dilated, and hypertrophy of the connective tissue may give rise to papillomatous growths on the vocal cords. The mucous glands, the posterior surface of the epiglottis, the false cords, and the sinuses of Morgagni are liable to become enlarged and dilated, and give the surface a granular appearance (granular laryngitis).

Chronic irritation often leads to hypertrophy of the squamous epithelium, which gives the affected spots a whitish appearance, and the condition sometimes spreads to parts that are normally covered with ciliated epithelium. The hypertrophy is most apt to affect the vocal cords, and may be combined with papillomatous thickening of the mucous membrane (Art. 142). VIRCHOW and others describe the condition as *pachydermia laryngis*.

Croupous inflammation of the laryngeal mucous membrane is sometimes primary, sometimes secondary to inflammation in the neighbouring parts. It is most common in connexion with diphtheria, small-pox, typhoid, and cholera. It may also result from the inhalation of irritating gases or hot vapours, or from the presence of foreign bodies. The interior of the larynx is covered with a white or yellowish more or less coherent false membrane, or only with white curdy flakes. These are sometimes readily detachable, sometimes more firmly adherent, especially when the affected part is provided with stratified squamous epithelium and papillae. The thickest and most adherent membranes are those that appear in diphtheria.

The false membranes consist partly of fibrinous filaments and meshes enclosing pus-corpuscles, partly of lustrous homogeneous flakes. When they are stripped off the underlying mucous membrane appears red, raw, and denuded of its epithelium.

True diphtheritic inflammation, with extensive sloughing and gangrene of the mucous membrane of the larynx, occurs most frequently in connexion with diphtheria and typhoid fever, though it is rare even in these diseases. Not uncommonly small necrotic foci are formed in the connective tissue underneath the croupous false membrane covering the epiglottis.

Oedema of the glottis is a more or less intense swelling of the laryngeal mucous membrane, due to dropsical saturation of the mucosa and in a greater degree of the submucosa. The swelling is usually greatest over the posterior surface of the epiglottis, the aryteno-epiglottic folds, and the false vocal cords, the submucosa of these parts being exceptionally loose in texture. The oedema may be so great in these situations as to occlude the opening into the larynx.

Oedema of the glottis may be acute or chronic. The former variety is due to inflammatory exudation, and occurs chiefly as a complication of catarrhal, croupous, or diphtheritic inflammation, as well as in the neighbourhood of syphilitic, tuberculous, and cancerous ulcers, and of submucous and perichondritic abscesses. It may also accompany suppurative inflammations of the pharynx, thyroid gland, and cervical connective tissue. In accordance with its mode of origin oedema of the glottis is often unilateral or confined to a single one of the parts just enumerated.

Chronic oedema is usually the result of venous engorgement from cardiac disease, pulmonary emphysema, compression of the cervical veins, etc. It is generally symmetrical and limited to the posterior surface of the epiglottis and the aryteno-epiglottic folds, though it may in less degree affect the vocal cords. Chronic inflammatory conditions of the larynx, such as ulcers or perichondritis, are also apt to give rise to inflammatory oedema of a somewhat chronic kind.

Phlegmonous inflammation of the larynx is an acute seropurulent and fibrino-purulent infiltration of the submucosa and mucosa, whose seat is generally the same as that of acute oedema. Suppuration of the tissues succeeds the infiltration, and **abscesses** are formed in the submucosa and mucosa, which on rupturing give rise to ulcers. When the inflammation extends down to the cartilages, purulent perichondritis is set up (Art. 242). Phlegmonous abscesses are apt to burrow in among the cervical muscles, or break into the pharynx and oesophagus. When the pus is evacuated the cavity may close up and heal by cicatrisation.

Phlegmonous laryngitis sometimes accompanies croupous, diphtheritic, and gangrenous inflammation, and tuberculous or syphilitic ulceration. In other cases it starts from inflammation of the perichondrium, pharynx, or tonsils, or from some acute traumatic lesion. The forms of laryngitis that are associated with typhoid fever, scarlatina, and pyaemia occasionally issue in suppuration.

Typhoid fever is frequently accompanied by a catarrhal laryngitis, marked by epithelial desquamation, ecchymosis, and superficial erosions, and by linear fissuring of the mucous membrane, especially about the edges of the epiglottis. Not uncommonly the posterior surface of the epiglottis, the anterior wall of the larynx, and the vocal cords are covered with a branny and more or less adherent fur, consisting of necrotic epithelium, leucocytes, micrococci, and bacilli. Sometimes also on the true and false vocal cords there are ulcers the floor and edges of which are beset with various forms of bacteria.

Less frequently than such non-specific ulcerations of the laryngeal mucosa in typhoid patients we meet with whitish diffuse swellings, or miliary nodules, due to dense cellular infiltration of the membrane. EPPINGER regards these as specific typhoid lesions, analogous to those that occur in the intestine. They appear mainly at the base of the epiglottis, on the false vocal cords, on the inner aspect of the arytenoid cartilages, and about the anterior attachment of the vocal cords. Disintegration of the infiltrated tissue gives rise to erosions with raised borders, closely resembling intestinal typhoid ulcers.

Both the specific and the non-specific ulcers are liable to extend in breadth and depth, ultimately setting up inflammation of the perichondrium (perichondritis), with necrosis and suppuration, which not infrequently causes extensive destruction of tissue, and necrosis of the affected cartilages.

The laryngitis of **small-pox** is characterised by the appearance on the reddened mucous membrane of minute whitish punctiform spots, or small lenticular nodules. According to EPPINGER, the former are due to cloudy swelling and granular degeneration, the latter to cellular infiltration, of the epithelium. As the infiltrated parts break down small round ulcers (Fig. 400) are produced. Sometimes a branny coating consisting of dead epithelium and pus-corpuscles or a coherent croupous membrane covers over the affected part. With these changes haemorrhages are associated in the case of haemorrhagic small-pox, and in the later stages small abscesses sometimes form in the connective tissue. Large perichondritic abscesses, with necrosis of cartilage, are however much less common.

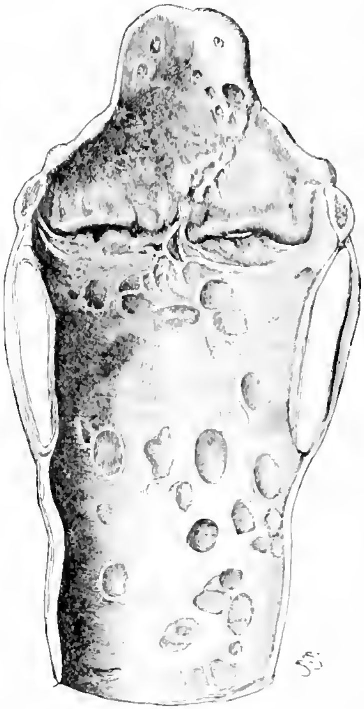

FIG. 400. VARIOLOUS ULCERS IN THE LARYNX AND TRACHEA.
(*Natural size*)

Scarlatina usually gives rise only to catarrhal laryngitis, seldom to the croupous or diphtheritic form, and the like is true also of measles and typhus. When **diphtheria** affects the larynx, it for the most part induces only croupous inflammation.

References on Laryngitis (see also Arts. 183 and 237).

Küttner: Laryngeal oedema and submucous laryngitis *V. A.* 139 **1895**
Landgraf: Affections of the pharynx and larynx in typhoid fever *Charité-Ann.* xiv **1889**
Posner: Cornification (pachydermia) of the mucous membrane *V. A.* 118 **1889**
Rheiner: Ulceration in the larynx *V. A.* 5 **1853**
Steudener: Histology of croup in the larynx and trachea *V. A.* 54 **1872**
Wagner: Diphtheritis and croup of the pharynx and air-passages *A. d. Heilk.* vii **1866**
Weigert: Croup and diphtheritis *V. A.* 70 **1877**

239. **Tuberculous laryngitis** is a very common complication of pulmonary tuberculosis; it also occurs independently, but this is rare. In the former case the specific infection of the larynx is doubtless effected by means of the bacilli contained in the sputum from the lungs. The process begins, after infection has once taken place, with the development of small sub-epithelial infiltrations projecting somewhat above the surface as greyish nodules. These rapidly become caseous and break through the epithelium, giving rise to small ulcers (Fig. 401 *a*) whose edges are not much thickened. This is simple tuberculous ulceration. In other cases the proliferation becomes more active and the cellular infiltration extends, with the result that sub-epithelial granulomatous tissue is formed, which encloses typical tubercles and assumes the appearance of irregular protuberances of various sizes rising from the surface of the mucous membrane (Fig. 402 *a b*). This is known as the hypertrophic form of laryngeal tuberculosis. Sooner or later caseation and disintegration set in, the epithelial covering is destroyed, and ulceration ensues.

Secondary changes presently appear in the form of disseminated patches of inflammatory infiltration in the mucosa and the submucosa, in the perichondrium, and sometimes also within the mucous glands or more rarely between the laryngeal muscles. These patches coalesce into larger masses of granulomatous tissue containing tubercles, and later on undergo caseation. This is most apt to occur in the perichondrium of the various cartilages.

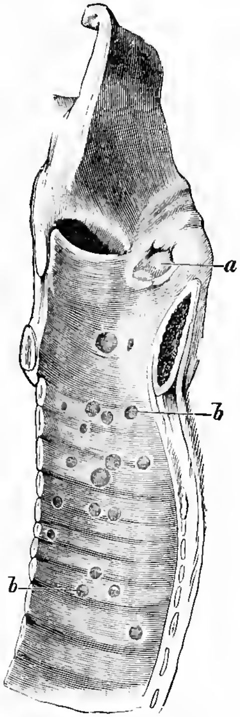

Fig. 401. Tuberculous ulceration of the larynx (*a*) and trachea (*b*).

(*Sagittal section through the larnyx and trachea*)

a deep-seated ulcer on the vocal process of the arytenoid cartilage

Larger tuberculous granulations are very commonly met with on the posterior surface and the edges of the epiglottis (Fig. 402 *b*), or on the anterior and posterior walls of the larynx (*a*). In the vocal cords, on the other hand, disintegration usually sets in before granulations of any size have time to develope.

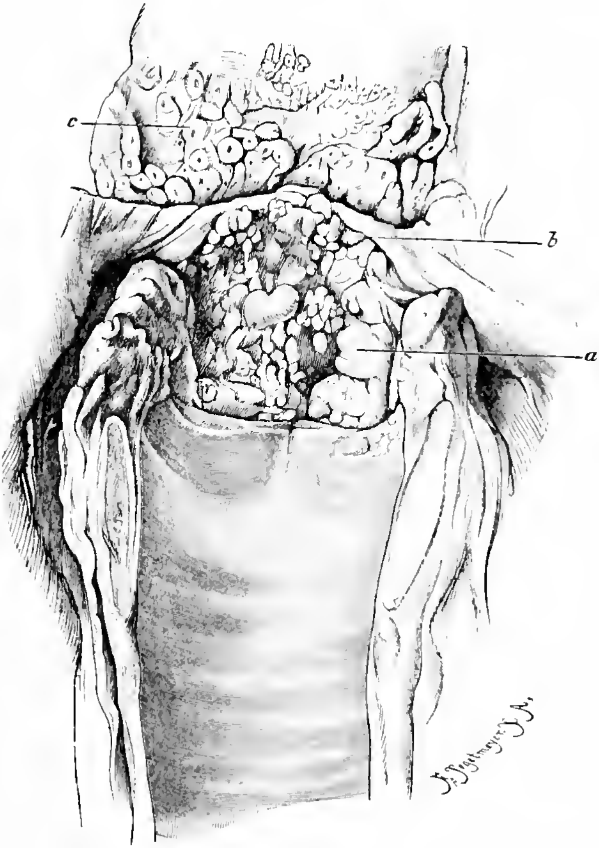

FIG. 402. HYPERTROPHIC TUBERCULOSIS OF THE LARYNX.

(*Trachea opened longitudinally from behind*)

a b papillary and nodose growths on the front and side wall of the larynx and posterior surface of the epiglottis

c enlarged follicular glands of the tongue

There is however no invariable rule in the matter, the extent of the tuberculous infiltration and of the subsequent ulceration varying greatly in different cases.

Tuberculous ulceration is always accompanied by more or less intense catarrh. As a result of the ulcerative process, oedema of the glottis and phlegmonous inflammation or even necrosis of the cartilages are sometimes induced.

Lupus of the nose and of the pharynx occasionally spreads to the larynx, and gives rise to nodulated and papillary outgrowths, and to ulcers with infiltrated edges. Local healing of the affected parts gives rise to cicatrices that usually cause more or less deformity.

References on Tuberculosis of the Larynx.

Biefels: *D. A. f. klin. Med.* xxx **1882**

Chiari and Riehl: Lupus *V. f. Derm. u. Syph.* **1882**

Garrè: Primary lupus of the glottis *D. med. Woch.* **1889**

Heinze: *Die Kehlkopfschwindsucht* Leipzig **1879**

Heryng: *Die Heilbarkeit der Larynxphthise* Stuttgart **1887**

Korkunoff: Genesis of tuberculous ulcers of the larynx *D. A. f. klin. Med.* xliii **1889**

Mackenzie: Pathol. histol. of laryngo-tracheal phthisis *Arch. of Med.* New York **1882**

Schech: Tuberculous laryngitis *Volkmann's klin. Vorträge* no. 230 **1883**

240. The first symptoms of **syphilitic laryngitis** may be those of a simple erythema and catarrh, though the accompanying infiltration of the mucous membrane is often extreme.

At a later stage deep erosions appear, which extend to the underlying structures, and whose floor and edges are densely infiltrated. Owing to the increased inflammatory infiltration, prominent greyish-white or reddish patches are formed on the surface of the mucous membrane (papular syphilitic laryngitis), which either ulcerate or if the infiltration is re-absorbed undergo resolution and disappear.

These erosions or ulcers, which are due to the disintegration of the infiltrated mucous membrane, vary much in extent and in depth. The floor of the larger ulcers is covered with a grey film; when this is removed the characteristic whitish exudation appears beneath. The epiglottis, the vocal cords, and the posterior wall of the larynx, are the most frequent seats of ulceration. In rare cases the greater portion of the interior of the larynx is denuded and the cartilages laid bare.

A second form of syphilitic ulceration of the larynx is due to the breaking down of gummata; these are usually seated in the submucosa, and are not due to direct infection from the pharynx. They are most common in the epiglottis (Fig. 403 *a*) and vocal cords, and may be so large and numerous as to obstruct or occlude the larynx.

Small gummatous nodes may be re-absorbed, but the large ones usually soften in the centre and break through into the larynx, giving rise to deep flask-shaped ulcers with infiltrated edges (Fig. 403 *a b*). The infiltration and the ulceration sometimes extend into the laryngeal wall, and cause perichondritis and necrosis of the cartilage: in this case the inflammation often takes on a suppurative character.

The syphilitic process may come to a standstill at any stage, the ulcers healing by cicatrisation. If the healing is delayed, considerable portions of the larynx, such as the epiglottis or the vocal cords, may be entirely destroyed. The greater the loss of substance, the larger is the cicatrix, and the distortion of the parts due to its contraction is often extreme. The cicatrix is white and hard; sometimes indeed the cavity of the larynx is constricted to a narrow and tortuous passage. The vocal cords occasionally become coherent, or the glottis is encroached on by protuberant bands of scar-tissue.

The islands of mucous membrane lying between the cicatrices are often thrust or bulged out in the process, and if they become inflamed and infiltrated or hyperplastic they take the form of outgrowths (Fig. 403 *b*) and papillomatous or polypous excrescences (*c*) which still further obstruct the air-passage.

Leprosy likewise gives rise to small nodules in the larynx, which coalesce into larger nodes and diffuse thickenings. The subsequent ulceration, cicatrisation, and contraction are apt to cause very great distortion of the parts, which is similar in appearance to that caused by syphilis.

In **glanders** disseminated inflammation is set up, which is characterised by the formation of sub-epithelial cellular nodules. These break down and ulcerate, and in this way extensive destruction of the mucous membrane takes place.

Rhinoscleroma also affects the larynx and trachea (Art. 157).

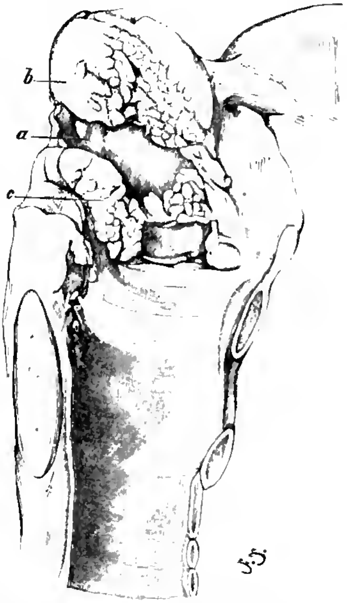

FIG. 403. SYPHILITIC ULCERATION OF THE LARYNX.

(*Sagittal section through the larynx and trachea: natural size*)

a ulcer
b thickenings and papillary growths on the epiglottis
c thickenings and papillary growths on the left laryngeal wall and the false vocal cord

References on Syphilis, Leprosy, and Glanders of the Larynx (see also Art. 237).

BERGENGRÜN: Leprosy of the larynx *A. f. Laryng.* II **1894**
BOLLINGER: *Art.* Glanders *Ziemssen's Cyclop.* III (and supplement) New York **1881**

GERHARDT and ROTH: Syphilis *V. A.* 21 **1861**
HAUFF: *Die Rotzkrankheiten* (glanders) *beim Menschen* Stuttgart **1885**
LANG: *Vorles. über Path. u. Therap. d. Syphilis* Wiesbaden **1885**
LEWIN: Syphilis *Berl. klin. Woch.* **1881**, and *Charité-Annalen* VI **1882**
SCHECH: Syphilis *D. A. f. klin. Med.* XX **1887**
THIN: Leprosy *B. M. J.* II **1884**
VIRCHOW: *Die krankhaften Geschwülste* II

241. Hyperplastic **mucous polypi** of the larynx are not common; but now and then we meet with polypous thickenings of the vocal cords, whose structure is exactly similar to that of the mucous membrane.

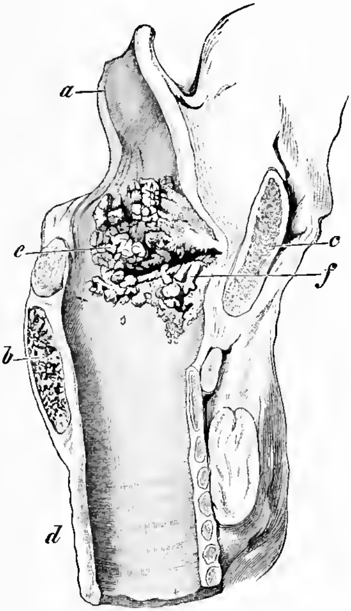

FIG. 404. FIBROUS PAPILLOMA OF THE LARYNX.

(*Natural size*)

a epiglottis
b ossified cricoid cartilage
c thyroid cartilage
d trachea
e f papilloma

Papillary or villous growths are much more common, and are described as **fibrous papillomata** or papillary fibromata (Fig. 404). Some of them are of inflammatory origin, others appear to be non-inflammatory. They generally grow from the true vocal cords, and sometimes extend over a considerable area. They take the form of compact nodulated tumours, or of branching papillary growths or cauliflower excrescences, which are sometimes described as *condylomata acuminata*. These papillomata have a fibrous framework consisting of simple or branched papillae, often infiltrated with round-cells, and sometimes permeated by wide blood-vessels, the papillae being covered by highly hypertrophic stratified squamous epithelium. Such overgrowth of the fibrous tissue and epithelium is included by some authors (VIRCHOW) under the term **pachydermia** of the larynx, while the papillary excrescences are spoken of as laryngeal warts.

Nodose **fibromata** are also most frequently met with on the vocal cords. They have broad or narrow bases, are smooth or warty, and are usually of the size of a lentil or small pea, though sometimes they are as large as a hazel-nut. Some are pale and bloodless, others red and vascular; some are hard, others soft.

Lipoma and myxoma are very rare. **Sarcoma** is somewhat more frequent; it generally resembles a nodose fibroma, but is rather softer. Chondromata have been more than once described: they start from the cartilages and form small knotty growths.

Primary **carcinoma** is most apt to arise about the vocal cords and the laryngeal sinuses. It may originate either in previously healthy tissue or in structures which have been modified by inflammatory changes (as in lupus). It takes the form of nodose or papillary growths (Fig. 405) or of diffuse infiltrations, which break down and leave ragged ulcers with an uneven floor. The ulceration is usually accompanied by purulent inflammation. The swelling and the destruction of tissue is sometimes very extensive, going far beyond the limits of the larynx. Secondary cancerous growths are more common, extending into the larynx by continuity from the oesophagus, pharynx, or thyroid gland: they reach this situation either by breaking through its walls, or else by growing downwards into it from the pharynx.

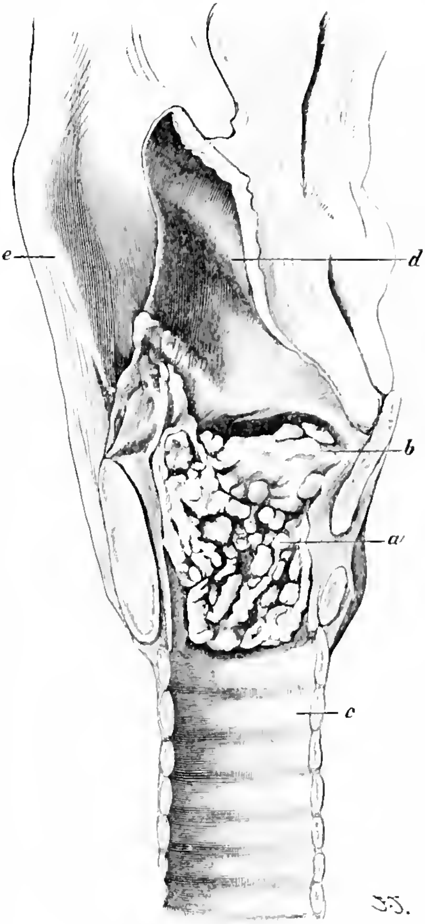

FIG. 405. CANCER OF THE LARYNX.

(*Sagittal section of the larynx and the trachea: natural size*)

a	cancerous growth
b	left vocal cord
c	trachea
d	left half of the epiglottis
e	pharyngeal wall

A few cases of **adenoma** have been noted, the growth taking the form of an irregular nodose tumour. Laryngeal tumours composed of tissue resembling the thyroid gland in structure have been described by VON ZIEMSSEN, BRUNS, and PALTAUF.

Cysts due to retention of secretion in the mucous glands are usually met with in the laryngeal sinuses and about the epiglottis, but they are not very common.

Of the **parasites** of the larynx, besides the specific and other bacteria already mentioned, we need refer only to *Oïdium albicans* and *Trichina spiralis*. The thrush-fungus gives rise to the characteristic white films; the *Trichina* lodges in the laryngeal muscles. Now and then round-worms (*Ascaris lumbricoides*) find their way into the larynx, and give rise to attacks of dyspnoea.

According to P. Bruns, out of 1100 tumours of the larynx 602 were papillomata, 346 fibromata, 73 mucous polypi, and 27 cysts. Seventy-six per cent. of the tumours were situated on the true vocal cords or at their anterior attachment.

References on Tumours of the Larynx.

Asch: Chondroma *New York Med. Journ.* **1884** (with references).
Baumgarten: Laryngeal carcinoma with tuberculosis *Arbeiten a. d. path. Inst.* Tübingen II **1894**
Bergengrün: Verruca dura laryngis *V. A.* 118 **1889**
Bruns, P.: *Die Laryngotomie zur Entfernung intralaryngealer Neubildungen* Berlin **1876**; Enchondroma *Beiträge von Bruns* III Tübingen **1888**
Bruns, V.: *Neue Beobachtungen über Kehlkopfpolypen* Tübingen **1873** and **1878**
Butlin: *Malignant disease of the larynx* London **1883**
Burow: *Berl. klin. Woch.* 13 **1876**; *Laryngoskop. Atlas* Stuttgart **1887**
Cervelato: Cysts *Lo sperimentale* **1881**
Chiari: Enchondroma *Wien. med. Jahrb.* **1883**; Pachydermia laryngis *Fortschr. d. Med.* IX **1891**
Crone: Carcinoma following lupus *Arbeiten a. d. Inst. v. Baumgarten* II **1894**
Elsberg: Angioma *Arch. of Med.* **1884**
Fränkel, B.: Cancer of the larynx *D. med. Woch.* **1889**
Hopmann: Warty tumours of the respiratory mucous membranes *Volkmann's klin. Vorträge* no. 315 Leipzig **1888**
Jurasz: Cysts *D. med. Woch.* **1884**
Küttner: Pachydermia laryngis *V. A.* 130 **1892**
Morell Mackenzie: *Growths in the larynx* London **1876**
Moure: Cysts *Gaz. des hôp.* **1880**
Paltauf: Thyroid tumours in the larynx and trachea *Ziegler's Beiträge* XI **1892**
Polewsky: Pachydermia laryngis *Fortschr. d. Med.* IX **1891**
Putelli: Chondroma of the larynx *Wien. med. Jahrb.* 84 **1889**
Schroetter: Lipoma *Monatsschr. f. Ohrenheilk.* **1884**; *Die Krankheiten d. Kehlkopfes* **1887**
Schwartz: *Des tumeurs du larynx* Paris **1886**
Sommerbrodt: Cysts *Breslau. ärztl. Zeitschr.* **1880**
Störk: Hypertrophy of the mucous membrane *Wien. med. Woch.* **1878**
Virchow: Pachydermia laryngis *Berl. klin. Woch.* **1887**
Werner: *Papillome d. Kehlkopfs* Heidelberg **1894**
Ziegler: Amyloid growths in the tongue and larynx *V. A.* 65 **1875**

242. The laryngeal cartilages are apt in old age to undergo certain physiological changes, which take the form partly of fibrillation, splitting, and disintegration, and partly of transformation into spongy osseous tissue. The process corresponds in details to the bony metaplasia or pathological ossification of the other cartilages of the body.

This softening and ossification also occur as a morbid change at an earlier age, especially in cases of chronic laryngitis. The transformation into bone always begins in the deeper parts of the cartilages and thence extends towards the surface.

Bile-pigment is sometimes deposited in the cartilages in cases of jaundice, and urates in gout.

The most important affection is however the inflammation of the perichondrium referred to as **laryngeal perichondritis.** It is usually a secondary affection, occurring in connexion with suppurative and ulcerative disease, tuberculous and syphilitic inflammation, and with carcinoma; but it also appears as an independent affection in pyaemia, small-pox, and severe cases of typhoid fever. Sometimes it originates in the decubital necroses met with in aged and debilitated bed-ridden patients at the posterior aspect of the cricoid cartilage, and due to the persistent pressure of the larynx on the vertebral column. Perichondritis may also be set up by mechanical violence.

The inflammation is usually purulent, but tuberculous, caseous, and indurative varieties are met with. It is nearly always limited to some part of the cartilaginous framework of the larynx, most commonly to parts of the cricoid and arytenoids. The accumulated exudation lying on the surface of the cartilage gives rise to more or less marked swelling of the parts, and presently portions of the cartilage become necrosed. When the perichondritic abscess bursts either outwards or inwards the cartilaginous sequestrum may be exfoliated and extruded. Abscesses bursting inwards usually give rise to inflammation of the bronchi and lungs, those bursting outwards to perilaryngeal suppuration.

After the abscess is evacuated and the dead cartilage removed, the wound may heal by granulation and cicatrisation. When the loss of substance is large, much contraction and distortion ensue. Smaller defects caused by injury or inflammation are filled up with fibrous tissue, actual regeneration of the lost cartilage taking place only to a very slight extent. So too in fracture of the cartilages by external violence, or in wounds due to operation (laryngotomy), repair takes place by means of new fibrous tissue, not of cartilage.

Now and then cartilaginous excrescences or **ecchondroses** make their appearance, and in cases where the cartilages have already become ossified **exostoses** have been described. They are usually found about the articulations, but are nearly always very small, not exceeding the size of a pea. A few instances of still larger growths are however on record.

References on Changes in the Laryngeal Cartilages.

BRIEGER: *Z. f. klin. Med.* III **1881**
GERHARDT: *D. A. f. klin. Med.* XI **1873**
LEWIN: Laryngeal perichrondritis *Charité-Annalen* XII **1887**
MORELL MACKENZIE: [illegible] London **1871**
SCHOTTELIUS: *Die Kehlkopfknorpel* Wiesbaden **1879**

CHAPTER LXXIX

THE TRACHEA

243. **Malformations** of the trachea are not common. In acephalous monsters the tube is at times entirely absent, the larynx and lungs being sometimes present, sometimes not. Occasionally we meet with cases of abnormally short trachea, and of atresia or narrowness of this or of one of the main bronchi. As a result of imperfect separation of the air-passage from the alimentary canal, there is occasionally a persistent communication between the trachea and the oesophagus, usually a little above the bifurcation of the former. When the two ends of the communicating passage become closed, it is transformed into a mucous cyst lined with ciliated epithelium.

Not infrequently some of the rings of the trachea are wanting; and in other cases they are abnormally coherent, or subdivided, or multiplied. The bifurcation may take place at an abnormally high level, or the first (epiarterial) branch of the right bronchus may arise directly from the trachea, which consequently gives off three main bronchi instead of two. Furthermore the trachea may give off a supernumerary branch, lying above the ordinary epiarterial branch of the right bronchus, and passing to the apex of the upper lobe of the right lung. According to CHIARI there often arises from the right main bronchus, above the normal epiarterial branch, a second branch which really belongs to the latter though it has been displaced on to the trunk of the main bronchus. This second branch is sometimes still further displaced so that it appears to arise from the trachea, and CHIARI states that it occasionally persists in a rudimentary form as a congenital diverticulum of the right wall of the trachea. Lastly we may have persistent remnants of the branchial clefts opening into the trachea, giving rise to the so-called cervical fistulae.

Acquired **dilatations** of the trachea are not very common; though we occasionally meet with cylindrical, fusiform, or sacculate dilatations, due probably to respiratory pressure, when expiration is obstructed and the tracheal wall more yielding than usual. Circumscribed sacculate dilatations are commonly situated on the posterior aspect of the tube.

Stenosis of the trachea is in general caused by compression from without; more rarely it is due to structural changes in the tube itself. Goitre and other tumours of the neck, peritracheal abscesses, and aneurysms of the aorta may be the cause of compression; cicatrices and other hyperplastic formations may give rise to obstruction from within.

Compression may be unilateral or bilateral. When it is very chronic it may induce atrophy of the cartilages, or lead to their transformation into fibrous tissue: it is however worthy of remark that sometimes no degenerative change is observed even when the compression has been extreme.

Perforation of the trachea, apart from mechanical injury, is most frequently due to cancerous and sarcomatous ulceration of the oesophagus or thyroid gland, and to aortic aneurysm, peritracheal abscess, or suppurating lymph-glands; it is much less commonly caused by ulceration within the trachea. In cases of aneurysm, the thinned-out wall of the sac pushes in the interannular spaces: the like effect is observed also in the case of carcinomatous and sarcomatous growths and of inflamed goitrous tumours.

Foreign bodies that become impacted in the trachea as a rule speedily set up inflammation and ulceration.

Wounds of the trachea are repaired by cicatricial tissue. Regeneration of cartilage takes place only to a very slight extent.

References on Malformations and Deformations of the Trachea.

D'AJUTOLO: A trachea with three bronchi *Mem. d. reale Accad. d. Scienze di Bologna* VI **1885**

CHIARI: A new type of malformation of the trachea *Ziegler's Beiträge* V **1889**; Duplicate epiarterial lateral branch of the right main bronchus *Prager Z. f. Heilk.* X **1889**; Congenital diverticulum of the right main bronchus *Prag. med. Woch.* **1890**; Trifurcation of the trachea *ibidem* **1891**

EPPINGER: Morbid anatomy of the larynx and trachea *Klebs' Handb.* Berlin **1880**

GERHARDT: *D. A. f. klin. Med.* XI **1873**

ROSE: Stenosis by compression *A. f. klin. Chir.* XXII **1877**

STILLING: Ciliated cysts of the anterior mediastinum *V. A.* 114 **1888**

244. The **inflammations** of the trachea present but few special features, and are frequently associated with inflammatory affections of the larynx. Catarrh is sometimes due to non-specific irritation: sometimes it is a complication of infective diseases such as measles, small-pox, whooping-cough, influenza, syphilis, etc. Laryngitis or bronchitis usually accompanies this latter form. Croupous inflammation is most common in diphtheria, and is characterised by the formation of a white fibrinous false membrane. Diphtheritic sloughing of the mucous membrane is not common.

Miliary **tuberculosis** of the tracheal mucous membrane is rare. Chronic tuberculosis is more frequent; it gives rise to subepithelial cellular infiltrations which afterwards break down and form ulcers of various sizes (Fig. 401 b). Sometimes these ulcers extend to the deeper structures, laying bare and partially destroying (by perichondritis) the cartilaginous rings. In rare cases the greater part of the mucous membrane is destroyed by ulceration.

Syphilis produces lesions resembling those of the larynx; indeed it frequently extends from the latter downwards, though it may appear in the trachea independently. In this case it is usually deep-seated, and is often associated with syphilis of the bronchi. The syphilitic inflammation not infrequently gives rise to extensive destruction of the trachea, extending even to the cartilaginous structures: the resulting cicatrices often cause by their contraction very remarkable distortion and stenosis of the tube, which may be beset in every direction with fibrous bands. The edges of the syphilitic ulcers are sometimes the seat of papillary excrescences partly covered with stratified squamous epithelium.

After tracheotomy fungous **granulations** sometimes spring from the internal wound, and seriously obstruct the trachea.

Primary tumours of the trachea are rare. Fibroma, sarcoma, chondroma, osteoma, adenoma, and carcinoma have been observed. Secondary growths due to extension from tumours of the oesophagus or thyroid gland are more common. Multiple detached osteophytes, in the shape of slender spicules and plates, are not very infrequent. They are seated in the mucous membrane, and are generally disseminated over the whole trachea.

Cysts arise from retention of secretion in the mucous glands. They are usually situated on the posterior wall and are sometimes as large as a walnut; as a rule they protrude into the space between the trachea and the oesophagus. Eppinger asserts that the mucous glands are liable to be distended with air forced into them through their ducts.

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CHAPTER LXXX

THE BRONCHI

245. The morbid changes affecting the larger bronchi in general correspond closely to those of the larynx and trachea. There are however certain peculiarities connected with them, arising partly from their anatomical structure and partly from their more intimate relation to the lungs.

Hyperaemia and **anaemia** of the bronchial mucous membrane have no very distinctive characters. **Haemorrhages** take the form of small ecchymoses of the mucous membrane, or of more copious effusions that mingle with the bronchial secretion. These are due to disturbance of the circulation, or to morbid changes in the vessels or tissues. In haemophilia, whether congenital or acquired, and more rarely in catarrhal inflammations, the haemorrhage is sometimes much more copious, and may partially fill the bronchi; while extensive suffusions appear in the mucous membrane. When the menses are suppressed 'vicarious' haemorrhage from the bronchi sometimes occurs. The blood effused into the tubes may be aspirated into the lung, and simulate pulmonary haemorrhage.

The commonest of all bronchial affections is **bronchitis.** In catarrhal bronchitis (Fig. 406) the mucous membrane yields a mucous ($f\,f_1$), serous, purulent, or mixed (Fig. 407 *a b*) secretion. The mucus so abundantly secreted in the acute stage comes partly from the lining epithelium, the cells of which undergo mucoid change (Fig. 406 $b\ c\ c_1$) and then discharge their contents or are thrown off themselves (*d*); and partly from the mucous glands of the bronchial wall (*n*). Drops of mucus may often be seen protruding from the orifices of these glands (*g*). The cellular elements contained in the bronchial secretion are pus-corpuscles and epithelial cells, which usually become mucoid and disintegrate (*d*).

When the secretion is very abundant, and is at the same time serous in character and with only a scanty admixture of cells, the affection has been called serous **bronchorrhoea**; when the secretion is more puriform the term **bronchoblennorrhoea** has been applied to it. Sometimes the secretion decomposes and becomes foul-smelling under the influence of putrefactive micro-organisms, and we have then foetid or **putrid bronchitis.** In all forms of bronchitis the mucous membrane is more or less densely infiltrated with cells (Fig. 406 *l o p*): this is most marked however in the purulent (Fig. 407 c c_1 d) and putrid forms, in which the infiltration extends even to the peripheral layers of the bronchial wall (*d*) and the peribronchial tissue (*e*). In the purulent form

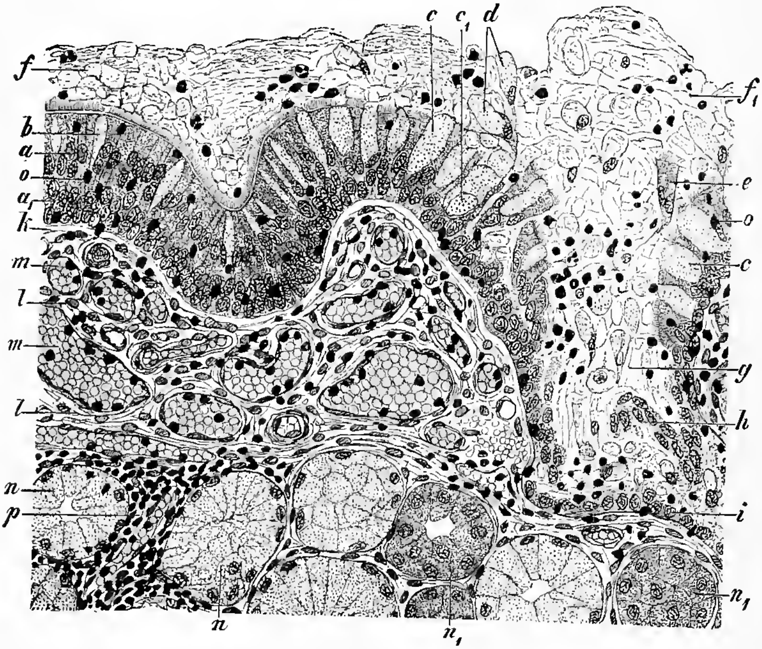

FIG. 406. RECENT CATARRHAL BRONCHITIS.

(*Preparation hardened in Müller's fluid and alcohol, stained with aniline-brown, and mounted in Canada balsam:* × 120)

a	ciliated cells
a_1	deep layers of cells
b	goblet cells
c	cells that have undergone extreme mucoid change
c_1	mucoid cells whose nucleus has undergone a similar change
d	desquamated mucoid cells
e	desquamated ciliated cells
f	deposit consisting of mucus-droplets, and
f_1	of mucus-filaments and pus-corpuscles
g	excretory duct of a mucous gland filled with mucus and cells
h	desquamated epithelium of the excretory duct
i	persistent epithelium of the duct
k	swollen hyaline basement-membrane
l	connective tissue of the mucosa somewhat infiltrated with cells
m	distended blood-vessels
n	mucous glands filled with mucus
n_1	acini of mucous gland without mucus
o	migratory cells in the epithelium
p	cellular infiltration of the connective tissue of the mucous glands

of catarrh the infiltrated epithelium is apt to be detached in some places (Fig. 407 c_1), and when the inflammation persists for some time undergoes mucoid change and is cast off. As a result of this process the smaller bronchi, which are normally invested with a single-layered epithelium (Fig. 407 c c_1), are denuded of their lining: those that possess a many-layered epithelium usually retain the cubical cells of the deeper layers for a considerable period. In severer forms of inflammation the epithelium occasionally becomes infiltrated with blood, and is thereupon loosened and shed. As recovery takes place, the lost epithelium is restored by a process of regenerative proliferation: sometimes this process begins during the actual course of the inflammation, and gives rise to the production of various irregular forms of epithelial cells.

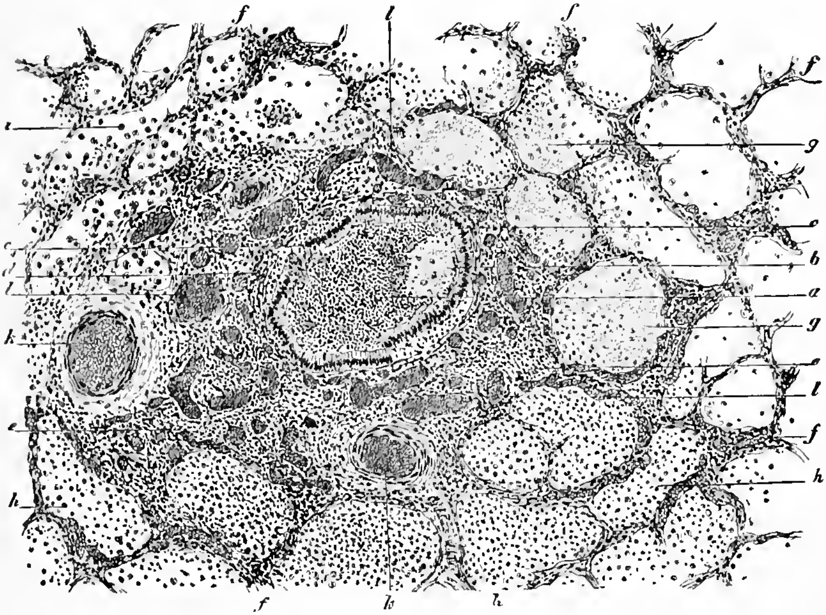

FIG. 407. PURULENT BRONCHITIS, PERIBRONCHITIS, AND PERIBRONCHIAL BRONCHO-PNEUMONIA.

(From a child fifteen months old: preparation hardened in Müller's fluid, and stained with haematoxylin and eosin: × 45)

a	purulent, and
b	mucous bronchial secretion
c	bronchial epithelium infiltrated with round-cells and partially detached (c_1)
d	bronchial wall infiltrated with cells and with its blood-vessels distended
e	peribronchial and periarterial connective tissue infiltrated with cells
f	interalveolar septa of the lung partially infiltrated with cells
g	fibrinous exudation in the alveoli
h	alveoli with highly-cellular exudation
i	alveoli with exudation containing few cells
k	transverse section of pulmonary arteries
l	bronchial, peribronchial, and interalveolar blood-vessels distended with blood

When bronchitis becomes chronic, atrophy of the component elements of the bronchial tube is a common result (Art. 248): in some places however hypertrophic proliferation of the connective tissue is set up (Art. 247). The epithelial lining of the tube often presents irregularities of structure, being here and there composed of one or of many layers of cubical or squamous polymorphous cells.

The causes of catarrhal bronchitis are numerous and diverse. Thus the affection may be due to the inhalation of irritating dust or to the aspiration of the contents of the mouth into the air-passages, while in other cases it is a symptom of one of the specific infective diseases, such as measles, diphtheria, whooping cough, influenza, or small-pox. The foetid form of bronchitis is most frequently associated with bronchiectasis or with gangrene of the lung, but it also occurs independently of these. Venous engorgement of the lungs favours inflammatory changes in the bronchi and impedes recovery.

Croupous bronchitis is usually an accompaniment of croupous tracheitis, and is almost always due to the specific virus of diphtheria: it may however be set up in other ways, as for example by the aspiration of irritating liquids from the mouth. In cases of croupous pneumonia there is always a certain amount of croupous inflammation of the smaller bronchi. The mucous membrane becomes covered over with whitish films whose thickness, except in the case of croupous pneumonia, is not great in any but the larger bronchi: in the smaller tubes mere specks and shreds of fibrin are formed, and as we pass to the finer bronchioles these gradually disappear and are replaced by catarrhal secretion.

There is also a chronic form of fibrinous or **plastic bronchitis**, in which from time to time firm coherent membranes form in the bronchi, and are coughed up as continuous tree-like casts of the ramifying tubes.

Diphtheritic and gangrenous inflammation with sloughing of the bronchial mucous membrane is rare. It is generally set up by gangrenous sputa coughed up from the lung or by the inhalation of powerfully-irritant matters. The inflammation thus induced is sometimes haemorrhagic, and patches of the mucous membrane and of the deeper structures of the bronchial wall become gangrenous and are thus destroyed.

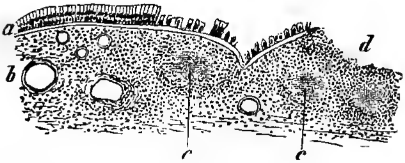

FIG. 408. TUBERCULOSIS OF THE BRONCHIAL MUCOUS MEMBRANE (× 25).

a epithelium
b fibrous tissue of the mucosa infiltrated with leucocytes
c tubercle
d margin of a small tuberculous ulcer

Tuberculous inflammation of the bronchi is a common accompaniment of pulmonary tuberculosis. It is usually most marked in the smaller tubes immediately connected with the diseased region, but in some cases it is diffused over the greater part of the bronchial system. Here as elsewhere the affection begins with the formation of grey cellular nodules (Fig. 408 *c*) which project somewhat above the surface. These caseate and break down, and in this way small ulcers (*d*) are formed whose floor and edges are usually covered with a whitish necrotic film, and are surrounded by a hyperaemic zone.

As the disintegration of the infiltrated margins of the ulcer advances, and the ulcer grows larger and coalesces with others, large irregularly-shaped erosions are at length formed, which sometimes extend to the cartilages of the bronchial wall. In the smaller tubes we frequently find the entire wall thus invaded and ulcerated away.

Syphilitic inflammation of the bronchi is not often seen: it presents the same appearances as in the trachea and larynx. Extensive loss of substance is occasionally caused by it; and as recovery takes place coarse puckered cicatrices are formed which are apt to give rise to notable contraction and distortion of the bronchi.

The tissue of the bronchial wall normally contains lymphoid elements, and in the larger (cartilaginous) bronchi these are in places aggregated into groups lying between the muscular coat and the cartilages: in this way lymphadenoid nodules are formed, which look not unlike tubercles.

CURSCHMANN has described under the name of **exudative bronchiolitis** a peculiar form of bronchitis in which tough hyaline or greyish or yellowish coagula are formed, 0·5–1 mm. thick and 1–2 cm. long, consisting of spiral or convoluted filaments and fibres, enclosing a variable number of cells. They are due to an exudative process affecting the bronchioles which, according to CURSCHMANN, is neither simple catarrh nor croupous inflammation. O. VIERORDT (*Berl. klin. Woch.* **1883**) describes similar coagula as occasionally met with in other inflammatory affections, such as croupous pneumonia. According to LEYDEN and LEWY, these coagula occur most frequently in bronchopneumonia, in which affection the epithelium is freely shed into the pulmonary alveoli and bronchioles.

In various forms of bronchitis, but especially in the croupous and exudative affections of the terminal bronchioles associated with **bronchial asthma** (LEYDEN, LEWY), the inflammatory exudation contains Charcot-Leyden crystals, in the form of slender acicular colourless octahedra. According to B. LEWY, these bodies are formed when there has been an extensive desquamation of epithelium, and do not occur in catarrh in which the desquamation is slight.

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246. **Stenosis** and **occlusion** of the bronchi are generally the result of inflammation. When the bronchial wall is infiltrated and the mucous membrane covered with exudations and secretions, the air-passage is always to a certain extent obstructed (Fig. 407 *a b c d* and Fig. 409 *a b*). As a rule this obstruction passes away, the morbid accumulations (mucus, pus, etc.) being removed by absorption and expectoration, while the swelling of the bronchial wall gradually goes down. Sometimes however the obstruction persists and becomes chronic.

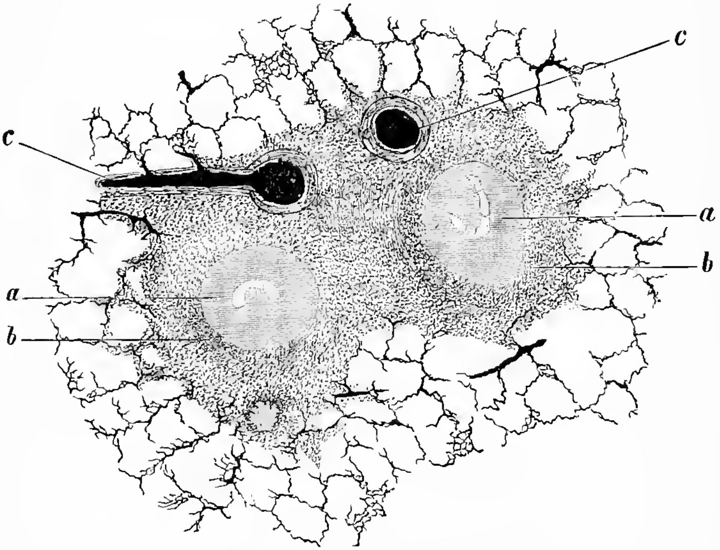

FIG. 409. TWO OCCLUDED BRONCHIOLES FROM A TUBERCULOUS LUNG.
(*Preparation injected with Prussian blue, and stained with ammonia-carmine* : × 25)

a caseous contents of the bronchioles
b bronchial wall and peribronchial tissue thickened and infiltrated with cells
c arterioles

Persistent obstruction of the bronchi may result from simple acute or chronic inflammation, but it is far more commonly due to tuberculous inflammation (Fig. 409 *a b*). This is owing to the fact that in tuberculosis we have not only thickening and infiltration of the bronchial wall, but also a secretion which contains many cells and little liquid. Accordingly in cases of chronic pulmonary tuberculosis there are always a certain number of obstructed bronchioles within the diseased region.

The contents of a bronchus thus occluded always become caseous (Fig. 409 *a*), so that on section it looks like a round encapsulated caseous node. Only when a considerable length of the tube is filled with caseous detritus and when the section cuts it more or less longitudinally, does it present the appearance of a cylindrical or elongated deposit. The boundary between the caseous contents and the bronchial wall is sometimes sharp and distinct, sometimes ill-defined. The former appearance is more characteristic of obstruction of the large bronchi, the latter of occlusion of the smaller bronchioles. The bronchial wall and the peribronchial connective tissue are generally thickened in the neighbourhood of the caseous deposit. The thickening after catarrh is oftenest simply fibroid, after tuberculous inflammation (Fig. 409 *b*) it is more fibro-cellular, the cells being in part denucleated, necrotic, and caseous.

The caseous contents of the tubes may after a time become calcified and form bronchial calculi.

Foreign bodies also may by impaction cause obstruction of the bronchial tubes. They give rise, according to their chemical and physical character, to indurative, purulent, or it may be putrid inflammation.

The cicatricial formations which follow upon destructive inflammation often by their contraction cause marked constriction and obstruction of the bronchi : this is well seen in syphilitic disease of the larger tubes.

In rare instances obstruction is caused by the growth of intrabronchial tumours.

Lastly, stenosis by compression from without is occasionally due to pulmonary tumours or inflammatory indurations, and at the root of the lung to enlarged lymph-glands, aortic aneurysms, and oesophageal tumours.

247. **Hyperplasia** and **induration.** After long-enduring bronchial catarrh thickening and papillary overgrowth of the mucous membrane is sometimes though not frequently observed. The change is never extensive, and is of small importance.

The induration and thickening of the entire bronchial wall, which results from certain forms of inflammation, is much more important. The change is most frequently observed in the neighbourhood of plugs of inspissated secretion, though it occurs also in unobstructed tubes, and sometimes extends over a considerable number of their ramifications. It may also affect the peribronchial fibrous tissue, and even extend to the contiguous parenchyma of the lung. Thus from what we may call endobronchitis is developed indurative mesobronchitis and **peribronchitis** (Fig. 410 *c d e*), with peribronchial lymphangitis.

Indurative peribronchitis may also arise from the like change (cirrhosis) commencing in the lung, the process being either of

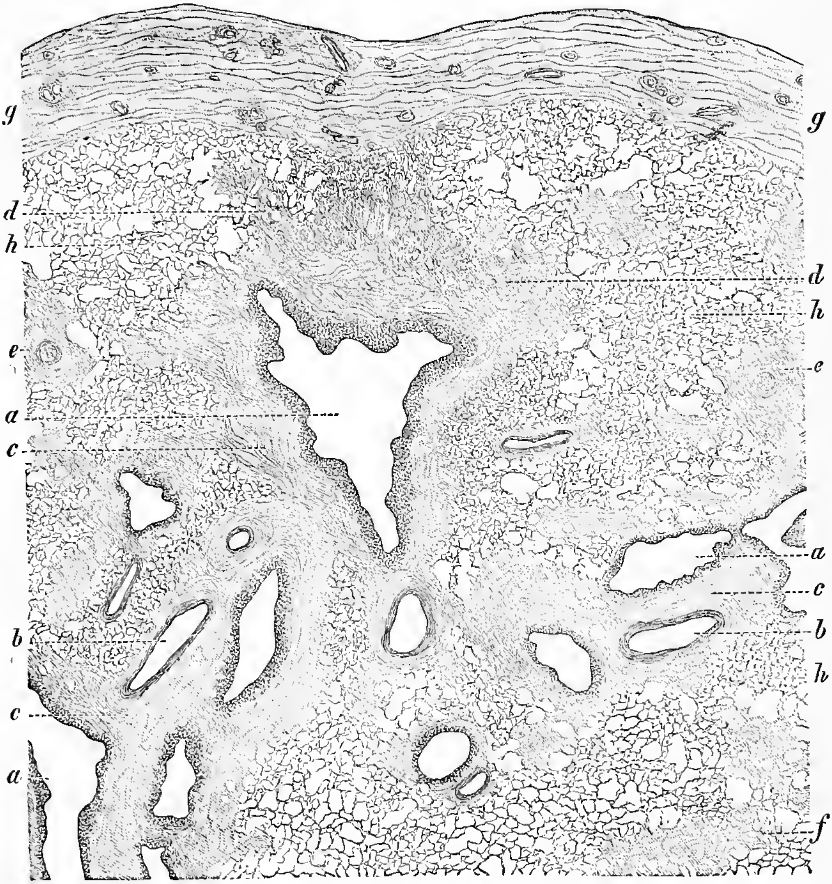

FIG. 410. FIBROID PERIBRONCHITIS.

(*Preparation stained with picro-carmine*: × 4)

a	bronchi, some of them dilated
b	arteries
c	thickened peribronchial fibrous tissue
d	fibrous bands radiating into the pulmonary tissue
e	bronchi blocked up with secretion and surrounded by indurated tissue
f	fibroid patches devoid of bronchi
g	thickened pleura
h	pulmonary tissue, partly emphysematous

the nature of direct extension or advancing gradually from the bronchioles of the indurated parenchyma by way of the lymphatics to the peribronchial fibrous tissue of the larger tubes. In like manner the inflammatory change may extend to the lymphatics of the peribronchial fibrous tissue from inflamed portions of the pleura or of the interlobular septa. In rare instances the induration extends from the fibrous tissue and lymph-glands at the root of the lung, and proceeds radially along the peribronchial structures.

The appearance of a bronchus thus thickened and indurated varies greatly, according to the way in which the process has been set up. If the lumen of the tube is unobstructed (Fig. 410 *a*) it looks on section like a thick-walled aperture sharply defined against the pulmonary tissue, or surrounded by radiating fibrous bands that project above the cut surface (*d*). If the tube is filled with inspissated secretion (Fig. 409 *a* and Fig. 410 *e*), the wall looks like a thickened capsule surrounding it. When the adjacent parenchyma of the lung is devoid of air, collapsed (Fig. 410 *d*), and indurated, there is no sharp line between the thickened bronchus and the altered lung.

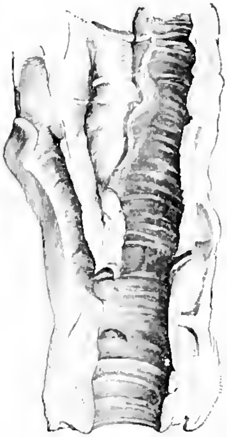

FIG. 411. ATROPHIC CYLINDRICAL BRONCHIECTASIS.

(*Dilated bronchus cut lengthwise, and showing prominent transverse ridges: natural size*)

Inflammations issuing in suppuration or in caseation affect the peribronchial tissues and lymphatics in the same way as the indurative variety: they often extend both widely and deeply. In tuberculous bronchopneumonia with caseation, caseous peribronchitis is always present, and in suppuration of the lung there is invariably a certain amount of purulent peribronchial lymphangitis. Of course the tubes immediately connected with the seat of disease in the lung are the first and most affected, but the process often spreads to the bronchi of other regions.

Peribronchitis being thus a secondary affection, and usually associated with bronchitic and pneumonic processes, it is generally accompanied by changes in the lung or in the pleura (Fig. 410 *g*). Indeed these latter changes are frequently the most apparent, and overshadow to a great extent the peribronchial lesions.

248. **Bronchiectasis**, or dilatation of the bronchi, results partly from increased internal pressure on the bronchial wall, partly from changes in its structure and in that of the surrounding pulmonary tissue.

The dilatation is either uniform (Fig. 411) and extending over one or more branches, or local and fusiform or saccular (Fig. 412): it may be single or multiple. Atrophic and hypertrophic forms of bronchiectasis are distinguished, in accordance with the character of the changes in the wall of the tube and in the peribronchial tis

Atrophic bronchiectasis is due to long-standing inflammatory affections, by which the strength and elasticity of the bronchial wall is considerably diminished, so that it yields to the internal pressure of the respired air. The development of this form is favoured by the presence of a temporary obstruction of the bronchus or of any impediment to full expiration. Such dilatations are usually cylindrical and are most marked in the lower lobes. When the wall yields unequally, the dilated tube appears sacculated, and its inner surface is irregularly corrugated with circular or oblique and sometimes anastomosing ridges (Fig. 411). These are simply the encircling muscular fasciculi of the bronchial wall, some of them enclosing elastic tissue, which to a certain extent retain their form in spite of the dilatation, while the mucous membrane bulges and yields between them.

The mucous membrane also is more or less atrophied and infiltrated with cells, the cartilages are often partially disintegrated and replaced by fibrous tissue, and the orifices of the mucous glands are dilated into small funnels. The epithelium is sometimes little altered; but in other instances it is seen that the columnar cells have become mucoid or detached, so that the surface is lined only with short cubical or club-shaped cells devoid of cilia.

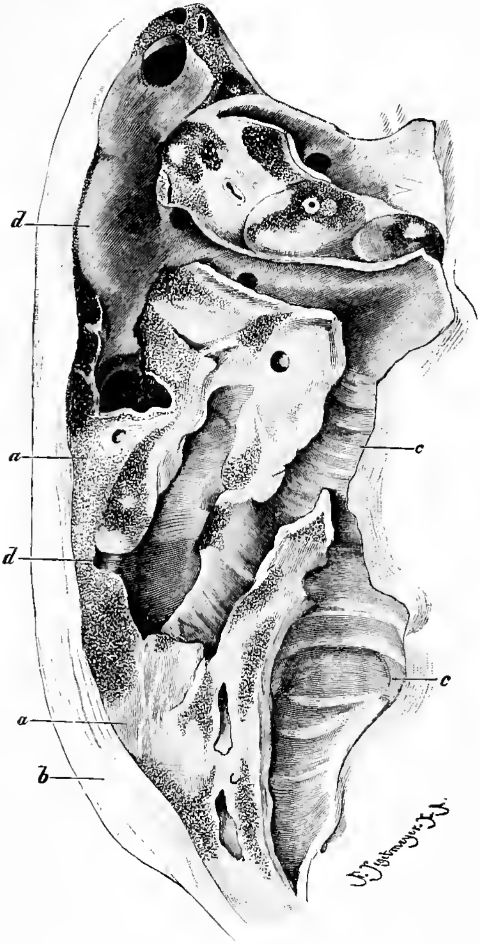

Fig. 412. Hypertrophic bronchiectasis.

(*Showing bronchiectatic cavities and fibroid induration of the pulmonary tissue: five-sixths of the natural size*)

- *a* indurated lung-tissue
- *b* thickened pleura
- *c* dilated bronchus
- *d* cavity communicating with a bronchus

This latter condition is especially marked where there is much catarrh.

Hypertrophic bronchiectasis (Fig. 412 *c*) is apt to supervene when some of the branches of an inflamed bronchus are impervious to air (*a*), so that the corresponding portion of the lung is collapsed and functionless. This is due not so much to any abnormal yielding of the wall of the bronchus as to the unequal distribution of the pressure in the lungs. The result is that on inspiration the air entering the bronchus is not uniformly distributed; and even if the neighbouring portions of the lung should dilate by way of compensation, as the thorax expands the air which rushes in is still unequally distributed, and bears unduly on the obstructed bronchus. It is easy to understand that inflammatory processes tend to weaken the wall of the tube, thereby allowing it to yield and favouring the dilatation. When the pulmonary tissue around a bronchus becomes shrunken and at the same time is adherent to the parietal pleura, it exerts traction on the bronchial wall, and gives rise to distortion and dilatation of the tube.

The hypertrophic bronchiectases (Fig. 412 *c*) brought about in the ways just enumerated may be fusiform or cylindrical, but they are more often saccular or entirely irregular in form. Sometimes in an indurated lung they are so numerous that the organ appears excavated in all directions. In very rare instances the dilatation behind a bronchial obstruction has the appearance of a regular cyst filled with mucus.

In these dilatations the mucous membrane undergoes changes similar to those just described in connexion with atrophic bronchiectasis. Papillary and polypous outgrowths from its surface are very rarely met with. The exterior layers of the bronchial wall and the peribronchial fibrous tissue, however, are frequently much thickened.

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249. **Ulceration** and **perforation** of the bronchial wall are due either to inflammation of the internal surface or to ulcerative affections of the surrounding tissues. Purulent, putrid, and tuberculous inflammations are those most apt to lead to ulceration and perforation from within. Suppuration is especially likely to occur

when septic matters are directly aspirated into the bronchi, or when the bronchial secretion undergoes putrefactive changes. The latter takes place chiefly in bronchiectases, where the secretion is apt to linger for a considerable time.

When perforation occurs and the originating inflammation extends to the surrounding parts, the peribronchial tissue and the adjacent lung-tissue become infiltrated, and according to the character of the primary affection undergo caseous necrosis or suppuration and gangrene. Caseous or purulent bronchitis thus issues in caseous or purulent peribronchitis, and a simple bronchiectasis is converted into an ulcerous bronchiectatic **vomica** (Fig. 412 *d*). The peribronchial excavation either lies beside the primary dilatation or surrounds it more or less completely.

The destruction of the bronchial wall is usually at first only partial, but in time it becomes complete, and the bronchus then appears to open into and terminate at the cavity. The walls of the cavity may appear gangrenous or caseous, infiltrated or indurated, according to the mode in which it has arisen and the point of time at which the examination is made. Its whitish or greyish liquid contents are puriform, putrid, or mingled with fragments of caseous detritus. The putrid contents include various forms of septic bacteria, and often spherules of leucin and needles of tyrosin and margarin.

The cavity in the first instance enlarges under the inspiratory air-pressure, and afterwards by the progressive disintegration of its walls: the enlargement takes place most rapidly when the process is suppurative or gangrenous, less rapidly when caseation accompanies the destructive inflammation, and least rapidly when the lung is already indurated by chronic inflammation.

Ulceration and perforation of the bronchi from without are generally dependent on suppuration, gangrene, or caseation of the parenchyma of the lung: they are extremely common. Caseous or suppurating lymph glands, peribronchial tumours, and aneurysms occasionally break through the bronchial wall.

When a bronchus is thus perforated, the broken-down tissue and detritus pass into its lumen, and are coughed up or aspirated into other parts of the lung. Air, on the other hand, may enter the excavation from the bronchus and thus cause it to dilate.

On **tumours** of the bronchi see Art. 273.

CHAPTER LXXXI

MALFORMATIONS OF THE LUNG

250. The **parenchyma** of the lung is composed essentially of the terminal bronchioles and alveoli and of the blood-vessels, together with a certain amount of connective tissue which unites the ultimate branches of the bronchi into lobules, and marks them off from one another. The transition from the air-tubes to the respiratory parenchyma is very gradual, the bronchial walls changing in structure by slow degrees, and ultimately becoming sacculated. The bronchi subdivide dichotomously into ever finer branches, and it is the finest terminal branches or bronchioles which go to form the respiratory parenchyma. At first the sacculations or alveoli occur singly (Fig. 413 *B*), and then in small groups on one side of the bronchiole. The tubes which are thus partially transformed into respiratory tissue are known as **respiratory bronchioles.** Each respiratory bronchiole divides into two or three smaller branches, which are surrounded on all sides (*B*) by alveoli and are known as **alveolar ducts.** The terminal alveoli are called **infundibula.**

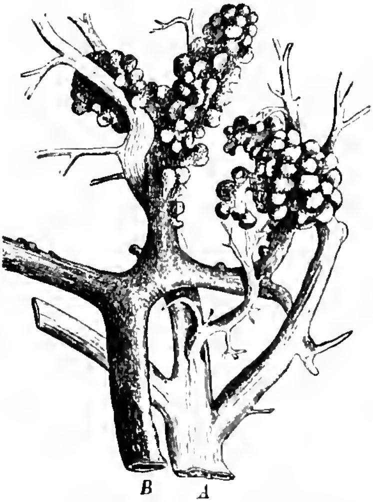

FIG. 413. TERMINATION OF A BRONCHIOLE (*B*) AND OF A PULMONARY ARTERIOLE (*A*).

(*Prepared by corrosion, magnified by a hand-lens*)

As the smaller bronchi pass into the respiratory bronchioles, they alter notably in structure. The cartilages disappear, and the epithelium is reduced to a single layer of low non-ciliated columnar cells, which ultimately assume the form of broad polygonal pavement-cells.

As the respiratory bronchiole changes to an alveolar duct, the modified columnar cells in turn disappear, and the epithelium takes the form of small nucleated granular-looking pavement-cells interspersed with larger hyaline plates, some with and some without nuclei. The muscular fibres of the bronchioles persist as annular bands surrounding the orifices of the lateral alveoli and of the terminal infundibula.

The epithelium of the alveoli is like that of the alveolar ducts. Their walls consist of a delicate fibrous membrane, surrounded by a close-meshed vascular network, and strengthened by scattered filaments and bundles of elastic tissue. They are devoid of muscular fibres.

The clustered alveoli belonging to each bronchiole are not quite contiguous, but are separated by spaces which are filled by other groups of alveoli and infundibula. The contiguous groups are bound together into lobules by connective tissue, which contains blood-vessels and lymphatics.

Malformations of the lung are on the whole not common. Partial or complete **hypoplasia** of one lung and partial **agenesis** of the parenchyma may occur, and is often accompanied by widespread cystic bronchiectasis.

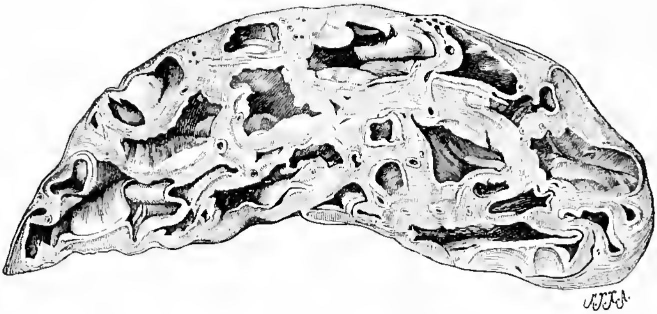

FIG. 414. AGENESIS WITH BRONCHIECTASIS OF THE LEFT LUNG OF AN ADULT.

(*Transverse section through the apex: the whole lung is abnormally small, and consists of vascular connective tissue devoid of alveoli, and pervaded by dilated and distorted bronchi: natural size*)

The lung developes from a single main tube. This tube grows into a vascular mass of cellular tissue, and as it grows divides and subdivides constantly, forming thus a multitude of ramifying terminal branches, on each of which are seated numbers of potential alveoli lined with cubical epithelium. After birth the air as it enters the lung dilates the alveoli. Absence of one lung is a very rare occurrence. In such a case the organ is represented by a small inconspicuous fleshy mass containing only a few ducts or rudimentary bronchioles. More commonly small circumscribed portions of the lung, limited it may be to the basal region of one lobe, remain undeveloped. The parenchyma then consists simply of cellular and highly-vascular connective tissue enclosing a variable number of ramifying bronchi, but no alveoli; or if any alveoli are present they are imperfectly developed. Other cases have been recorded in which though the lung was of some bulk, but still much smaller than normal, its parenchyma was found to consist essentially of cellular connective tissue with a few rudimentary bronchi, but no well-developed alveoli (Fig. 414).

These appearances are most commonly met with in still-born infants, or in those that have died shortly after birth. It is, however, worthy of note that such conditions are not necessarily incompatible with life, as they are occasionally discovered in the bodies of children of various ages, and even in adults (Figs. 414 and 415). When the malformation is restricted to a small region, the remainder of the lung developes and grows in such a manner as to render it capable of performing the necessary work of respiration. When the whole or the greater part of one lung is imperfectly developed, the condition known as **compensatory hypertrophy** supervenes in the sound lung; and cases have been recorded in which a single lobe has become so enlarged as to occupy the greater portion of the thoracic cavity, the heart being displaced towards the side of the undeveloped lung. In new-born infants the bronchi in the abnormally-developed portion of the lung are sometimes normal in appearance; but in other cases the tubes are dilated, and thus congenital fusiform and cystic bronchiectases may be found enclosed in the dense unexpanded lung-tissue. When the patient has lived for any length of time, the malformed portion of the lung (Fig. 414) is sometimes converted by progressive dilatation of the bronchi into a mass of tissue honeycombed with cysts varying in size from that of a pea to that of a hen's egg. In such cases the several cysts may either communicate by orifices of varying width with a more or less dilated main bronchus (Fig. 415), or they may appear entirely shut off from it and from each other.

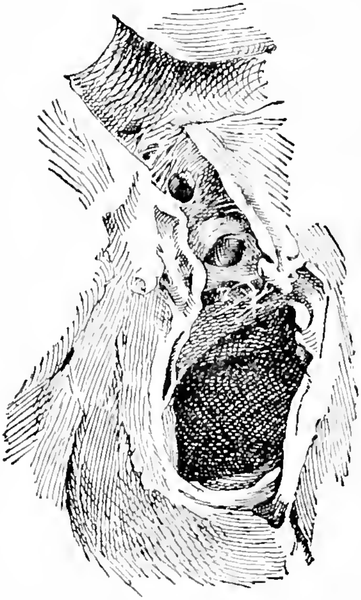
FIG. 415. SACCULAR BRONCHIECTASIS WITHIN DENSE UNEXPANDED LUNG-TISSUE.

(From the base of the right upper lobe of an adult)

The walls of the cysts are either thin and delicate, or firm and dense, and are lined by ciliated columnar epithelium. When it is the larger bronchi that have become cystic, the fibrous tissue forming their walls generally contains plates of cartilage. The fibrous tissue interposed between the cysts is more or less abundant.

So long as no secondary changes take place, the tissue of the anomalous region is not pigmented, even in the adult; and no adhesions to the costal pleura may be present. If the dilated main bronchus becomes the seat of inflammation which extends to the cysts, secondary changes supervene, such as pleural adhesions, induration and accumulation of secretion in the cysts, and even haemorrhage, the latter leaving behind it patches of brown pigmentation.

The commonest remaining anomaly, and one which has no functional significance, is excessive **multiplicity of the lobes.** As the result of some imperfection of development vesicular dilatation of a group of alveoli has in a few rare cases been observed: this might be described as congenital parenchymatous **emphysema.**

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FEUSTEL: The ultimate results of atelectasis *Inaug. Diss.* Kiel **1883**
FURST: *Gerhardt's Handb. d. Kinderkrankh.* III Tübingen **1877–83**
GRAWITZ: Congenital bronchiectasis *V. A.* 82 **1880**
GRUBER: Various anomalies *V. A.* 81 **1880**, 102 **1885**, 103 **1886**
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HELLER: The results of atelectasis *D. A. f. klin. Med.* XXXVI **1885**
VON KÖLLIKER: *Zur Kenntniss d. Baues d. Lunge* Würzburg **1880**
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PONFICK: Primary atrophy of the right lung *V. A.* 50 **1870**
SCHMIT: Agenesis of both lungs *V. A.* 134 **1893**
SHATTUCK: *Ziemssen's Cyclop.* (supplement) New York **1881**
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CHAPTER LXXXII

ATELECTASIS AND EMPHYSEMA

251. In the unborn child the lung is a compact structure; the alveoli exist potentially, but they are everywhere collapsed and airless. Only when respiration commences do the alveoli become distended by the incoming air into hollow vesicles, and the epithelial cells lining their walls are thus expanded and flattened.

If the expansion is imperfect, owing to occlusion of a bronchus or compression of a part of the lung, some of the lobules remain undistended and retain the dense fleshy consistence and the bluish-red or brownish-red tint of the foetal organ. This condition is known as **foetal atelectasis** or apneumatosis.

When a part of the lung which has once expanded in respiration becomes from any cause airless, it is said to be in a condition of **acquired atelectasis** or **collapse.** The condition may be due to compression, to obstruction of a bronchus, or to the presence of solid or liquid exudations in the alveoli. Compression of the lung is most commonly brought about by the collection of air or liquid in the pleural cavity, or by excessive elevation of the diaphragm; it may also be due to aortic aneurysm, spinal curvature, thickening and contraction of the pleura, distension of the pericardium, etc. The compression may be partial or total, and the resulting collapse accordingly more or less complete.

When the collapse affects the whole lung and is complete, the organ is usually retracted against the spinal column, and its tissue is dense, firm, and airless; its colour is generally pale-pink or in pigmented parts grey. Collapsed segments of the lung have a similar appearance, but there is often more blood in the parts and they have accordingly a redder colour.

When a bronchus or bronchiole is occluded by secretion or other cause, the corresponding segment always becomes airless after a time. LICHTHEIM states that the oxygen of the enclosed air is first absorbed by the blood, then the carbonic acid, and ultimately the nitrogen; the lung thereupon shrinking to its foetal condition.

As the collapsed part no longer expands or contracts with respiration, and as its capillaries are much folded and contorted, a certain amount of vascular engorgement takes place. The unexpanded tissue thus looks somewhat livid in tint, and appears retracted and sunken in comparison with the normal tissue around it.

Obstructive collapse is extremely common, and is indeed a usual accompaniment of inflammation of the smaller bronchi.

Post mortem, the lung looks mottled with livid retracted patches alternating with pink or reddish-white air-containing regions.

If foetal atelectasis persists, changes take place in the tissues of the affected part similar to those described in Art. 250 as the result of partial agenesis and hypoplasia, except that some alveoli lined with flattened epithelium are always present in the condensed tissue. It is difficult to determine whether these alveoli disappear in the course of time, inasmuch as their absence at a later stage may be attributable to partial agenesis *ab initio*. It is however possible and indeed probable that in some cases the alveoli are actually obliterated, the bronchi at the same time becoming more or less dilated.

When a part of the lung remains collapsed for some time, certain indurative changes in its tissues usually make their appearance. These changes are due to the proliferation of the pulmonary connective tissue, and they lead ultimately to the obliteration of the alveoli. The alveoli are however lined with epithelium, and this impedes the complete cohesion of their walls in the collapsed portion of the lung. Collapse may therefore exist for a long time without inducing the entire obliteration of the alveoli, which indeed probably occurs only when inflammatory changes supervene. For this reason morbid induration or **cirrhosis from collapse** must be looked upon as the result of coexistent or subsequent pneumonic or bronchopneumonic processes.

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CALMAN: Collapse *B. M. J.* I **1881**
DUNIN: Morbid anatomy *V. A.* 102 **1885**
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HELLER: Final condition of atelectatic portions of the lung *D. A. f. klin. Med.* XXXVI **1885**
LICHTHEIM: Experiments on pulmonary collapse *A. f. exp. Path.* X **1879**
SCHUCHARDT: Extreme chronic atelectasis of the left lung with compensatory hypertrophy of the right *V. A.* 101 **1885**
TRAUBE: *Gesammelte Beiträge z. Physiol. und Path.* Berlin **1871**
WEBER: *Beitr. z. path. Anat. d. Neugeborenen* Kiel **1852**

252. When the thorax is over-distended by forced inspiration, or when one part of the lung is pervious to air while another part is shut off, the pervious part becomes excessively inflated, and a condition which we may describe as **acute vesicular emphysema** is induced. This condition also occurs when on account of the swelling of the bronchial mucous membrane, or the accumulation of secretions in the bronchi, air can enter the alveoli in inspiration but cannot escape during expiration. The alveoli are not altered in structure but are simply over-distended. This condition is very commonly the result of bronchopneumonia. The over-distended lobules are pale and anaemic, and those that lie

immediately beneath the pleura project like little blebs above the level of the normal or collapsed portions.

When the pressure within an alveolus exceeds a certain amount its walls give way, and air enters the interalveolar tissue, and in particular the lymph-channels. This condition is called **inter-vesicular emphysema**. It is generally a result of bronchitis and bronchopneumonia accompanied by violent coughing, and is met with in children who have died of asphyxia during the course of these affections. It may also occur from over-energetic attempts to insufflate the lungs of still-born infants.

The alveoli of the anterior border of the upper lobe are the most apt to give way. The inflated vesicles are usually subpleural, and may be as large as a pea. Sometimes air passes from them under the pleura toward the root of the lung and into the mediastinal areolar tissue, occasionally even inflating the subcutaneous structures of the neck and thorax (subcutaneous emphysema).

When the alveoli are subjected to persistent or often-repeated distension, partial atrophy and yielding of their walls ensue, and two or more alveoli being thus converted into one, the pulmonary tissue is to that extent rarefied. This state is called **chronic vesicular emphysema** or simply emphysema. Its production may be facilitated by disorders of nutrition, such for example as accompany local inflammations or senile decay (senile emphysema). The lungs of many persons seem normally to possess but little power of resistence to over-distension.

The atrophy of the interalveolar septa begins at the point where they are thinnest, and is first manifested by the widening of the intercapillary spaces (Fig. 416 *a*) and the yielding or disappearance (*b*) of the fine elastic fibres of the alveolar wall. Holes and gaps next appear between the capillaries in the septa; they are at first very small (*b*), but soon enlarge (*d*). The over-stretched capillaries become impervious (*c*) and ultimately give way (*d*).

By the gradual extension of this process many of the septa and their capillaries at length disappear, the thicker fibrous bundles which surround the alveolar ducts being the last to go. The epithelium is passive throughout, but often shows signs of degenerative (generally fatty) change. Sometimes the tissue is inflamed and infiltrated, but this has nothing to do with the emphysema as such; it is simply a concomitant of the catarrh which so frequently affects patients suffering from emphysema.

Chronic emphysema may be due, like the acute variety, to persistent inspiratory over-distension of the lung-tissue. This occurs chiefly in cases where parts of the lung are collapsed and functionless (Fig. 430 *e*), and the neighbouring parts (Fig. 430 *g*) are accordingly over-distended. We might describe this as vicarious or **compensatory emphysema**. It is sometimes lobular, sometimes lobar, in its extension. The emphysematous lobules are inflated and their alveoli abnormally large.

On the other hand emphysema also results from persistent and violent expiratory efforts, in conditions which interfere with the egress of air from the alveoli, its ingress being unimpeded. This is the case in the important variety described as chronic idiopathic diffuse emphysema, or simply **general emphysema,** an affection which is very common in persons that are subject to chronic bronchial catarrh or recurrent expiratory dyspnoea (asthma), or are obliged to make violent expiratory efforts in connexion with their employment (glass-blowers, trumpeters, etc.).

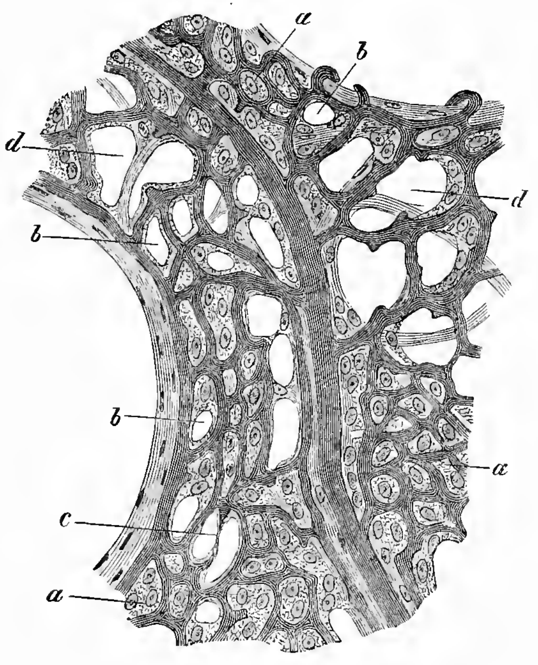

FIG. 416. CHRONIC VESICULAR EMPHYSEMA.

(*Injected preparation, stained with carmine, and mounted in Canada balsam:* × 200)

- *a* dilated intercapillary spaces with epithelial cells
- *b* gaps in the alveolar septa (EPPINGER'S primary dehiscence)
- *c* capillary in process of obliteration
- *d* larger gaps in the alveolar septa (EPPINGER'S secondary dehiscence) and in the capillary network

This form of emphysema extends over the whole lung, though it is usually most marked at the edges and apices of the lobes and at the base of the lung. When the lung is removed from the thorax it appears abnormally large, it feels soft and downy, its edges are obtuse and rounded, and the base is frequently studded with hemispherical bladder-like prominences. The air-vesicles are everywhere enlarged by the disappearance of the interalveolar septa, sometimes so much so that they look like little bladders ranging in size from that of a pea to that of a hen's egg. When the air is pressed out the tissue of the lung is seen to be diminished in bulk, while its edge collapses into a flaccid inelastic membrane.

The smaller vesicles (Fig. 417 *a*) are formed by the disappearance of the interalveolar septa belonging to a single infundibulum; the larger vesicles (*b*) by the disappearance of the partitions between adjacent infundibula.

When some of the vesicles in the general or in the local form are of exceptional size, we have what is called **bullous emphysema**. The air is usually not easily pressed out of the larger bullae.

The bronchi may be normal in calibre or dilated; in one form of chronic emphysema the dilatation and atrophy of the bronchi are so marked that they constitute an essential feature of the affection.

In emphysema a large number of capillaries are obliterated, and the area of distribution of the pulmonary artery being thus contracted, the resistence to the circulation through it is increased. Compensatory hypertrophy of the right ventricle of the heart is thus a frequent concomitant, while the pulmonary arterioles that remain are often visibly dilated.

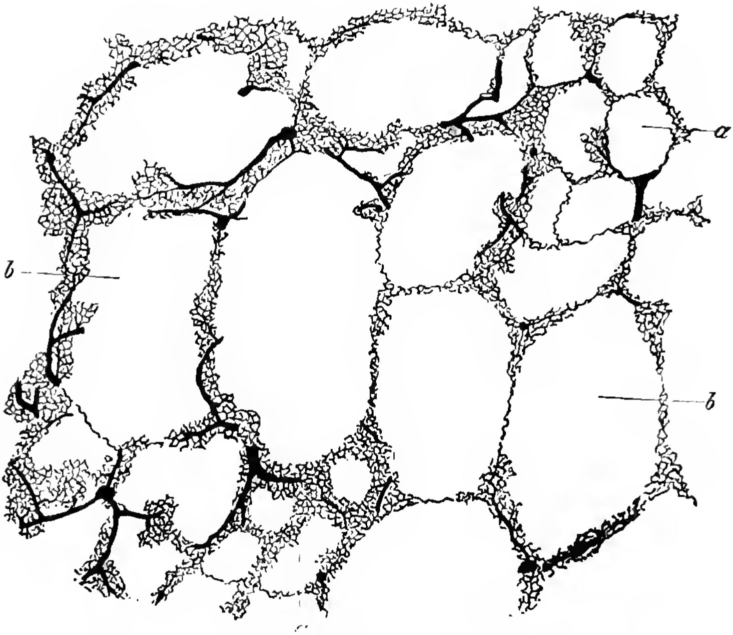

FIG. 417. RAREFIED PULMONARY TISSUE IN EMPHYSEMA.

(*Injected preparation* : × 20)

a simple infundibular vesicle produced by disappearance of interalveolar septa

b large bulla produced by coalescence of infundibular vesicles

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BIERMER: *Volkmann's klin. Vorträge* no. 12 Leipzig **1870**; Acute emphysema *Berl. klin. Woch.* **1886**
EPPINGER: *Vierteljahrsschr. f. prakt. Heilk.* IV **1876**
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CHAPTER LXXXIII

DISORDERS OF CIRCULATION IN THE LUNG

253. **Congestive hyperaemia** is in some cases due to diminution of the normal resistences to the arterial current within the lung. It may be induced directly by stimuli reaching the lung with the inspired air, as when irritating or irrespirable gases are inhaled or when the air is excessively hot or excessively cold: it may be induced indirectly through the blood, when that liquid is contaminated by toxic substances. Hyperaemia is also the first step to inflammation. Moreover, partial or collateral congestion of one part of the lung sometimes results from obstruction of an important arterial branch in another part.

Congestion of the lung, when it is not collateral or due to local textural or vascular changes (as in inflammation), extends uniformly over the whole organ. It is usually transient, and is very seldom fatal. In the fatal form of congestion (sometimes called **pulmonary apoplexy**) the lung appears swollen and abnormally firm, of a uniform dark-red colour on section, and containing but little air: the capillaries are everywhere distended with blood and encroach on the alveolar cavities. There are usually also some scattered extravasations of blood.

Engorgement or passive hyperaemia results from hindrance or obstruction to the outflow of blood through the pulmonary veins, or from causes tending to weaken the propelling forces: it is therefore very frequently observed on post-mortem examination of patients who have died slowly. Want of propulsive power becomes apparent as the right ventricle fails and the respiratory activity diminishes, while failure of the left ventricle prevents the proper outflow of the blood from the lungs. When air is hindered from entering the lungs on inspiration, blood is as it were pumped from the extra-thoracic vessels into the intrathoracic, and accordingly accumulates in the venous channels connected with the right side of the heart.

Obstruction to the outflow of blood from the lung is most frequently caused by incompetence or stenosis of the mitral valve, but the same effect is also indirectly produced by obstructive increase of pressure at the aortic orifice and in the aorta itself.

Local passive hyperaemia of the lung occurs chiefly in connexion with local failure of the respiratory function (as in partial collapse), or with obstruction and occlusion of some of the pulmonary vessels. When the engorgement is great the affected parts become livid or purple in colour. The wide-spread hyperaemia of the pulmonary vessels that makes its appearance in dying patients is most marked in the dependent portions of the lung, and is accordingly described as **hypostatic engorgement.** When owing to mitral valvular disease the outflow of venous blood from the lungs is persistently impeded, and the right heart is thereby forced to greater activity, the blood-pressure within the pulmonary vessels is increased, and these accordingly become permanently dilated. The dilatation shows itself most in the capillaries, which are visibly distended and project from the walls of the alveoli. The lung in consequence becomes firmer in consistence, and the condition is therefore described as **induration from engorgement.** In many places the tissue is condensed, or shows signs of inflammation and proliferation, though these are due less to the hyperaemia than to the recurrent haemorrhages that are apt to take place in such lungs (Arts. 255 and 256).

Anaemia of the lung may be due to general anaemia. When it is merely local, it is usually dependent on compression or excessive inflation of the part, or on obstruction of some of its vessels. After death the blood generally drains from the anterior portions of the lung to the deeper and posterior portions, so that the former are apt to look anaemic.

254. **Oedema** of the lung is a condition indicated by the escape of serous liquid more or less abundantly mingled with air into the alveoli, bronchioles, and frequently into the bronchi also. When the cut section of the lung is squeezed a clear liquid escapes, which may or may not be mixed with bubbles of air.

This condition is very commonly met with *post mortem*, sometimes affecting both lungs and in other cases confined to one lobe of one lung, or it may be to a few lobules. The pulmonary tissue may be either anaemic or hyperaemic; in the latter case the liquid filling the alveoli is often tinged with blood (sanguinolent oedema).

The oedematous liquid generally contains few formed elements, though the proportion of these depends on the originating cause of the oedema. Desquamated alveolar epithelium (Fig. 418) is the chief and often the only solid constituent. In adults the desquamated epithelial cells generally contain black granules of inhaled coal-dust; sometimes they are literally crammed with carbonaceous particles.

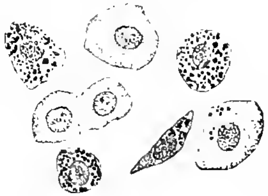

FIG. 418. DESQUAMATED ALVEOLAR EPITHELIAL CELLS, CONTAINING SOME CARBONACEOUS PARTICLES, FROM A CASE OF PULMONARY OEDEMA.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and carmine*: × 300)

The most frequent form of pulmonary oedema is that known as **passive oedema.** This form is of course associated with passive hyperaemia, and so appears in the most dependent portions of the lung. The exuded serous liquid is often tinged with blood, the coexisting engorgement tending to promote the extravasation of red blood-corpuscles.

Another common variety of pulmonary oedema is distinguished by the absence of any signs of engorgement. In this form the oedema involves the whole lung, and not merely the dependent portions, and the pulmonary tissue is often deficient in blood or entirely bloodless. It appears to be due to some alteration of the vessel-walls that causes them to be abnormally permeable, and is probably referable to the action of septic poisons elaborated during the course of the disease from which the patient suffered. We may therefore describe it as **toxaemic oedema.** Some of the blood-vessels in these cases contain hyaline coagula (Thoma), and occasionally (as when death has been preceded by some process involving disintegration of the tissues) fat-emboli also.

A third variety is the **inflammatory oedema** that generally accompanies intense pulmonary inflammations, or appears independently in the course of septicaemic or pyaemic infections. In this form of oedema the desquamation of pulmonary epithelium is very free, and the transuded liquid is rich in albumen: accordingly when the lung-tissue is hardened in alcohol or in Müller's fluid granular precipitates of albumen are apt to make their appearance in the section.

Inflammatory oedema of the lung cannot be sharply distinguished from the toxaemic form, and its characters pass without abrupt transition into those associated with certain forms of catarrhal inflammation. When the primary inflammation is severe the exuded liquid contains leucocytes, and sometimes fibrin also, and so becomes more or less turbid. The lung-tissue itself may on section appear either pale or reddened.

References on Oedema of the Lung.

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Löwit: The cause of pulmonary oedema *Ziegler's Beiträge* xiv **1894**
Mayer, S.: *Wiener Sitzungsber.* **1878**, and *Prager med. Woch.* **1880**
Thoma: Pulmonary oedema *Petersburger med. Woch.* **1889**, and *Arbeiten aus dem pathol. Institut d. Univ. Dorpat* **1889**
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255. **Haemorrhage** from the pulmonary vessels is of very common occurrence, and arises from a great variety of causes. In

the first place, haemorrhage is very frequently a result of venous engorgement. The quantity of blood which escapes is not usually so great as to cause firm haemorrhagic infarction, but it may lead to the formation of rather large dark-brown patches of infiltrated and airless tissue.

When a considerable amount of serous liquid transudes with the red blood-corpuscles, we have what is called **haemorrhagic oedema** of the lung. If the air is entirely displaced from the lung-tissue, so that it looks dark-red and not unlike a soft and very vascular spleen, the condition has been termed **splenisation** of the lung. It is most commonly a result of gradual cardiac failure preceding death: the blood, being no longer efficiently propelled, accumulates in the deeper parts of the lung and so gives rise to what we might describe as hypostatic haemorrhagic oedema. If as often happens inflammation supervenes in the engorged region, the process is termed **hypostatic pneumonia.**

Extravasation of blood is an exceedingly common accompaniment and consequence of chronic passive hyperaemia from mitral incompetence or stenosis, and of pneumonic and bronchopneumonic affections. In chronic engorgement the extravasations are irregularly scattered and of various sizes; they are ill-defined in extent and are dark-red in colour. Larger or smaller haemorrhages or their traces are nearly always found in the lung (Art. 256) after death from mitral valvular disease.

In recent acute inflammation the red corpuscles escape from the vessels with the inflammatory exudation, of which indeed they form a component part. In the later stages of the inflammation, when the pulmonary tissue breaks down, haemorrhage is usually due to the rupture of small or large blood-vessels whose walls have been previously softened or ulcerated. In the case of the larger arterial branches the vessel-wall usually yields and becomes dilated into a small **aneurysm** before actual rupture takes place. These aneurysms are most frequently observed on vessels which traverse or lie in the wall of ulcerous cavities. When they rupture more or less copious haemorrhage ensues, and the cavities together with the bronchi which open into them are flooded with blood.

Mechanical injury, like that caused by a bullet or a broken rib, gives rise to bleeding from the lung whose amount depends of course on the nature and extent of the wound.

In copious haemorrhage such as follows the rupture of an artery, blood passes into the bronchi and is coughed up (**haemoptysis**). Some of the blood may be aspirated from the bronchi into their smaller branches and the corresponding alveoli. In this way blood-soaked patches exactly resembling haemorrhagic infarcts are formed; usually however their number and distribution and the circumstances in which they occur enable us to determine their true nature.

In somewhat rare cases pulmonary haemorrhage is referable to a congenital or acquired haemorrhagic diathesis, as in haemophilia or scurvy; or to infective diseases, like scarlatina, typhoid, and small-pox; or lastly to cerebral disease, especially such as causes disturbance of the respiratory function. In the latter case the bleeding may be very considerable, whole segments of the lung becoming airless and saturated with blood.

The most marked form of haemorrhagic infiltration or **infarction** is that which follows thrombosis or embolism of a branch of the pulmonary artery. The infarct is usually subpleural, of a sharply-defined conical form with the base directed outward, and in the recent state dark brownish-red in colour and firm in consistence. The emboli come from the right side of the heart or perhaps from the systemic veins, and usually lodge at the bifurcation of the arterial branches (riding emboli). The characteristic extravasation appears to take place only when the artery is completely blocked, and the blood reaching the affected region from the neighbouring capillaries is wholly insufficient to maintain the circulation. These conditions are most likely to exist when the lung is already engorged (as in mitral incompetence or stenosis). When the circulation is well maintained, however, the initial disturbance caused by the embolism is speedily counteracted and no infarction is produced.

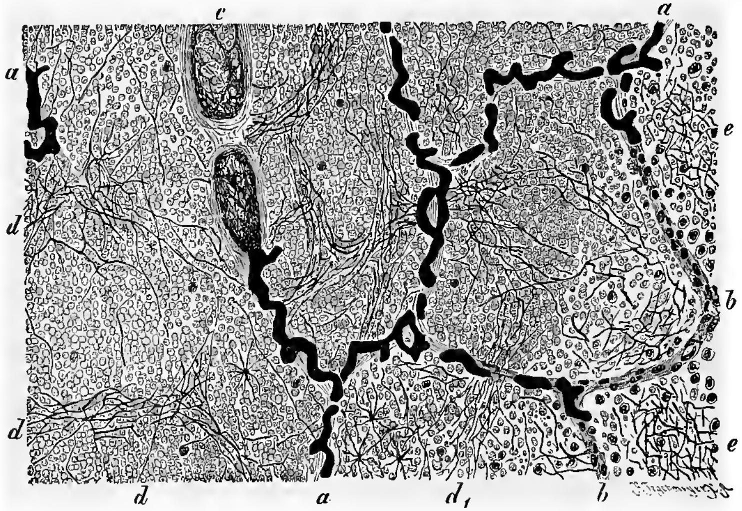

FIG. 419. MARGIN OF A RECENT HAEMORRHAGIC INFARCT OF THE LUNG.

(*Preparation hardened in Müller's fluid, stained with haematoxylin and eosin, and mounted in Canada balsam* : × 100)

a	denucleated alveolar septa containing capillaries filled with homogeneous thrombi stained dark-purple
b	nucleated septa
c	vessel with red thrombus
d d_1	alveoli filled with coagulated blood
e	alveoli filled with serum, fibrin, and leucocytes

Embolic haemorrhagic infarcts on section vary in size from that of a cherry-stone to that of a hen's egg, though occasionally they are much larger. They are distinguished from local extravasations due to engorgement mainly by their sharply-defined boundaries, and by the denser infiltration of the affected tissues. The blood that has escaped into the alveoli is coagulated (Fig. 419 d d_1), the lung-tissue within the region of the infarct is denucleated (a) and necrotic, and the contiguous parts after a time appear inflamed (e). The pleura over a recent infarct is smooth and glistening, but afterwards it becomes turbid and covered with a thin fibrinous film.

When the blood contains an excessive number of colourless corpuscles, as in leukaemia, the infarct is sometimes pink or yellowish-red in colour. This is due to the presence of leucocytes, which accumulate in great numbers in the pulmonary vessels.

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Cohnheim and Litten: The results of embolism of the pulmonary arteries *V. A.* 65 **1875**
Gerhardt: *Volkmann's klin. Vorträge* no. 91 **1875**, and *Clinical lectures* (New Syd. Soc.) London **1877**; *Handb. der Kinderkrankh.* III
Grawitz: *Virchow's Festschrift* (*Assistenten*) Berlin **1891**
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Klebs: *Schweiz. Arch. f. Thierheilk.* XXVIII **1886**
Küttner: The circulation in the mammalian lung *V. A.* 73 **1878**
Mögling: The formation of haemorrhagic infarcts *Ziegler's Beiträge* I **1884**
Virchow: *Gesammelte Abhandlungen* Frankfort **1856**
Zuckerkandl: Arterial anastomoses in the human lung *Wiener Sitzungsber.* LXXXVII **1883**

References on Pulmonary Haemorrhage in Cerebral Affections.

Brown-Séquard: *Lancet* I **1871**
Charcot: *Leçons sur les maladies du syst. nerveux* Paris **1875** (trans. by Sigerson) London **1876–89**
Jehn: *Cent. f. med. Wiss.* **1874**
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Pinel: *De l'hémorrhagie pulmonaire en rapport avec les lésions du cerveau* Paris **1876**

256. When the pulmonary alveoli contain liquid or blood, changes invariably take place therein which have for their object the removal of the abnormal contents. This is always ultimately accomplished provided the patient does not die in the meantime. Liquids are rapidly removed on the re-establishment of the normal conditions of circulation and respiration. Some of the red blood-corpuscles are taken up by the interalveolar lymphatics and so are carried off. Most of the corpuscles are however disintegrated in

the usual way: some are dissolved, while others break down into coloured or colourless granules and flakes, and yellowish or brownish deposits containing iron; and cells enclosing the detritus of the red corpuscles (pigment-granule cells, Fig. 420 *b*) speedily make their appearance within the alveoli. Some of these are desquamated epithelial cells from the alveolar walls, others are immigrant leucocytes, and others again in chronic cases are derived from the proliferous connective tissue of the lung. Not infrequently some of the alveoli are found to be entirely filled with such pigment-carrying cells.

Some of the cells containing disintegrated blood from the alveoli pass into the bronchioles and are removed with the sputum. As pulmonary haemorrhages are very common in connexion with the engorgement attending heart-disease, these cells are frequently observed in the sputum of patients suffering from cardiac lesions, and some writers have actually described them as 'heart-disease cells' (WAGNER, HOFFMANN, SOMMERBRODT). The term is not very appropriate, though it may perhaps be justified on grounds of clinical convenience. Another portion of the blood-pigment reaches the pulmonary lymphatics, where it may remain for long or be carried at once to the lymph-glands. After a time cells containing yellowish and brownish pigment-granules and flakes (Fig. 420 *c*) are found in the connective tissue of the

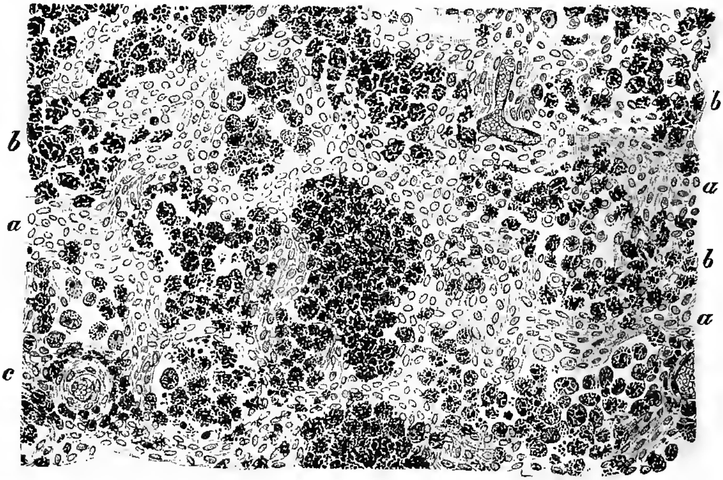

FIG. 420. PULMONARY INDURATION FROM ENGORGEMENT WITH HAEMORRHAGE, FROM A CASE OF MITRAL INCOMPETENCE AND STENOSIS.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin:* × 100)

a proliferous and thickened alveolar septa
b groups of granule-cells containing haemosiderin
c thickened circumvascular fibrous tissue containing pigment-granule cells

lung, giving rise to more or less permanent patches of ochreous or brownish pigmentation. In cases of frequently-recurrent haemorrhage, such as that associated with pulmonary engorgement from heart-disease, the process of absorption is liable to be accompanied by much fibrous hyperplasia (*a*), which gradually leads to thickening of the interalveolar septa, and here and there even to induration of the pulmonary tissue and obliteration of the

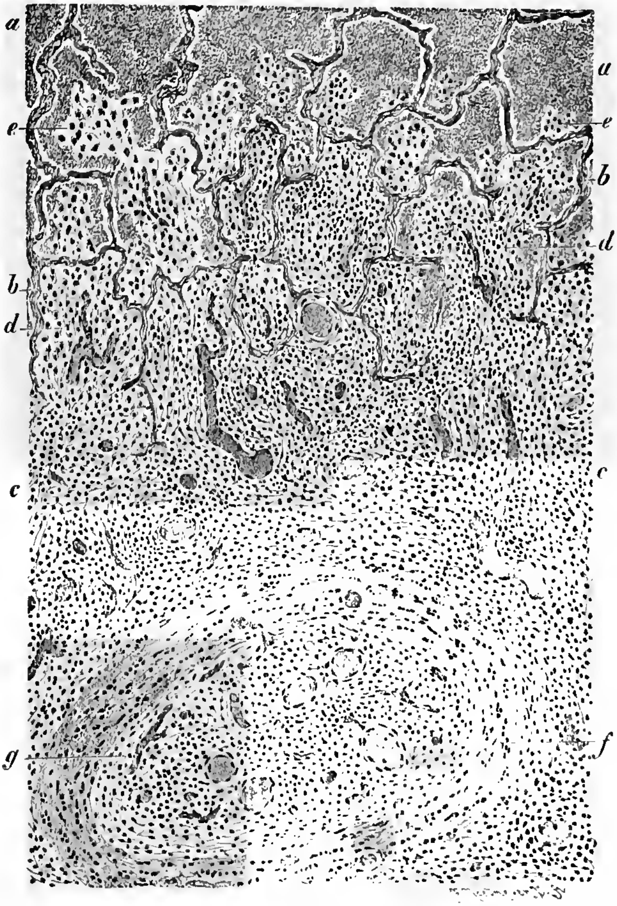

FIG. 421. MARGIN OF A CICATRISING INFARCT OF THE LUNG, WITH OBLITERATED ARTERIES.

(*Preparation hardened in Müller's fluid, stained with haematoxylin, and mounted in Canada balsam:* × 45)

a haemorrhagic extravasations converted into yellowish granular masses
b necrotic denucleated interalveolar septa
c new-formed fibrous tissue
de vascular granulations within the alveoli
f artery
g vascular fibrous tissue replacing an arterial embolus

alveoli. This process gives rise to a condition which, on account of the abundance of pigment contained in the thickened tissue, is known as **brown induration** of the lung.

When the part involved in a haemorrhagic infarction is small, and the tissue does not undergo necrosis and disintegration, the lung may return to its former condition in a few weeks. Repair is effected by the liquefaction and absorption of the extravasated blood, and a little pigmentation and thickening may thus be the only structural change that is left behind. When however the infarction is followed by necrosis of the damaged tissue, many weeks or months may elapse before the defect is made good by **cicatrisation**, provided the patient survives and no destructive infection of the part supervenes. The reparative process in this case consists in the resorption of the infarcted tissue, with the formation of granule-carrying cells and the production of new fibrous tissue. The formation of new tissue is most active at the zone of demarcation between the living and the dead portions, and all the constituent elements of the pulmonary tissue take part in it.

As proliferation thus takes place at the periphery of the infarct, granulation-tissue (Fig. 421 *c*) is formed and grows into the necrotic mass, eventually displacing and superseding it. Hence in the course of a few months even a large infarct may be replaced by cicatricial connective tissue, so that nothing remains but a scar-like condensation of the pulmonary tissue covered with thickened pleura. In some instances however decolorised remnants of necrotic tissue are left behind in the interior of the cicatrix.

The **cicatrix** resulting from these processes is relatively small, because compensatory expansion of the adjoining alveoli causes some of the space vacated by the removal of the necrotic tissue to be filled up; while the cicatrix itself is not altogether continuous, but still encloses a few residual alveoli and bronchioles.

During the process of repair, the embolus and the thrombi deposited on it are replaced by fibrous tissue (Fig. 421 *g*), the arteries either becoming entirely obliterated or merely receiving a certain amount of thickening on their walls and so becoming pervious again.

When the embolus producing the infarction contains at the same time infective micro-organisms capable of setting up suppuration or putrefaction, or when these reach the injured tissue with the inspired air, suppuration or gangrene of the lung may ensue (septic pneumonia, Art. 264).

References on the Resorption of Pulmonary Extravasations and on Changes in the Lung from Chronic Venous Engorgement.

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Buhl: Dilatation of the pulmonary capillaries *V. A.* 16 **1859**
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Hoffmann, F. A.: The significance of 'heart-disease cells' *D. A. f. klin. Med.* xlv **1889**
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Orth: Brown induration of the lung *V. A.* 58 **1873**
Perl-Lipmann: Experimental researches on pulmonary haemorrhage *V. A.* 51 **1870**
Sommerbrodt: Genesis and significance of the 'cells of heart-disease' *Berl. klin. Woch.* **1889**; Has blood effused into the air-passages a causal relation to pulmonary consumption? *V. A.* 35 **1866**
Zenker: *Beiträge zur path. Anatomie der Lungen* Dresden **1862**

CHAPTER LXXXIV

NON-INFLAMMATORY DEGENERATIONS OF THE LUNG

257. The non-inflammatory **degenerations** of the pulmonary tissue are of comparatively slight importance, and have no practical interest for the physician. Emphysema and senile atrophy form exceptions, but they have already been discussed in Art. 252.

Swelling, fatty degeneration, and desquamation of the alveolar epithelium accompany every copious transudation into the alveoli, inflammatory or non-inflammatory. The inhalation of deleterious substances also leads to manifold lesions of the epithelium, blood-vessels, and fibrous framework of the lung; but the changes so induced are usually of an inflammatory nature.

Among the degenerative changes due to disorders of nutrition, we may mention **fatty degeneration** of the epithelium and **amyloid degeneration** of the blood-vessels: the former occurs in emphysema and in poisoning by phosphorus and arsenic; the latter in conditions which lead to amyloid changes elsewhere. It is however to be kept in mind that the lung-tissue itself is rarely the seat of amyloid disease; it is the walls of the blood-vessels that are most apt to be affected.

Amyloid concretions (*corpora amylacea*) are occasionally seen in pneumonic exudations, in old haemorrhagic patches, in tuberculous foci, and in emphysematous portions of the lung.

Calcification of the fibrous tissue of the lung is rare, except in cases where it has been morbidly altered by antecedent inflammation. This change is generally associated with affections that involve extensive resorption of bony tissue in other parts of the body.

References on Calcification and Fatty Degeneration of the Pulmonary Tissue.

CHIARI: Calcareous deposits *Wien. med. Woch.* **1878**
CORNIL and BRAULT: Fatty degeneration after phosphorus-poisoning *Journ. de l'anat.* XVIII **1882**
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CHAPTER LXXXV

PNEUMONOCONIOSIS AND PULMONARY INFLAMMATIONS IN GENERAL

258. Most of the circumscribed inflammatory lesions of the lung are caused by the inhalation along with the inspired air of irritating substances, which set up pathological conditions in the parts in the lung-tissue where they are arrested. In this connexion, then, we have to consider diseases that extend from the ramifications of the bronchi to the parenchyma of the lung, and we may fitly include all such inflammatory affections under the head of local or **disseminated bronchopneumonia.**

The irritating substances may affect both the bronchi and the alveoli, and the resulting bronchitis and pneumonia are thus simultaneous effects of the same cause. In other instances the irritating substances are arrested only in the larger bronchi, and the inflammation so induced extends thence to the bronchioles and to the respiratory parenchyma; in this case the bronchopneumonia is really a secondary lesion.

Of such inhaled irritants **dust** in all its forms is by far the most common. We all inspire a certain amount of dust with the air of the street or of the house, while in certain occupations the amount of dust necessarily inhaled is very considerable. Workers in stone of all kinds, masons, bricklayers, potters, mill-stone workers, stone-cutters, and cement and porcelain makers, often inhale large quantities of mineral dust; workers in metal, such as grinders, gilders, braziers, type-founders, and moulders, inhale fine metallic particles; millers, colliers, coal-heavers, chimney-sweeps, bakers, cabinet-makers, rope-makers, cigar-makers, and workers in spinning and weaving mills, live in an atmosphere charged with dust of vegetable origin; brush-makers, upholsterers, barbers, cloth-dressers, and hat-makers breathe air containing animal dust; and glass-workers, street-sweepers, etc., dust of various other kinds.

A large proportion of the dust thus inhaled is caught in the air-passages, but some of it, especially in deep inspiration, is carried into the parenchyma of the lung. Many of the particles adhere to the walls of the alveoli, others are promptly conveyed into the lymph-channels communicating with the alveoli, and thence are carried by the peribronchial and interlobular lymphatics into the lymph-glands at the root of the lung.

When a considerable number of dust-particles reach the parenchyma of the lung, they set up a slight inflammation manifested by the emigration of white blood-cells from the vessels, and by the swelling, desquamation, and even proliferation of some of the alveolar epithelial cells.

The desquamated and extravasated cells take up the foreign particles (Fig. 418), sometimes in such abundance that they have fitly been termed **dust-cells.** Some of them are carried into the bronchioles and bronchi, and are then ejected with the sputum. A number of them are however conveyed into the lymphatics.

Within the lymph-channels certain kinds of dust, such as chalky particles, are dissolved. Insoluble dusts are either carried into the bronchial lymph-glands or are deposited in the walls of the lymphatics. This deposition takes place wherever lymphatics occur, namely in the interalveolar, interlobular, subpleural, pleural, circumvascular, and peribronchial fibrous tissues, especially in those parts where aggregations of lymphoid elements are normally met with. The particles lie either free in the tissues or enclosed in rounded, fusiform, or stellate cells.

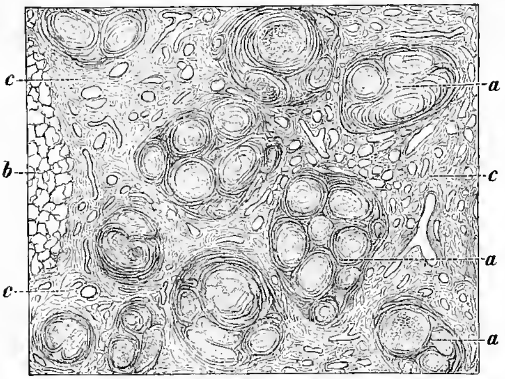

FIG. 422. SECTION OF A STONE-CUTTER'S LUNG, WITH BRONCHOPNEUMONIC FIBROUS NODULES.

(*Preparation hardened in alcohol, and stained with picro-carmine:* × 5)

a cluster of fibrous nodules
b normal lung-tissue
c indurated lung-tissue still containing bronchioles, vessels, and a few alveoli

Coloured dusts give rise to **pigmentation** of the lung, while the larger grains appear as sandy or gritty deposits.

Some kinds of dusty deposition, in particular those that give rise to easily-perceptible changes in the lung, have received special names, although all are included in the general term **pneumonoconiosis**. The most frequent as well as the best-known form is that due to the inhalation of soot or coal-dust, by which the lung becomes dark-grey or black in colour; it is variously described as **anthracosis** or *pneumonoconiosis anthracotica* (ZENKER). This form of pigmentation is extremely common; indeed it is seldom entirely absent in adult lungs, and is usually most marked about the apices.

A second form is the so-called **siderosis** or *pneumonoconiosis siderotica* (ZENKER), due to the inhalation of metallic dust;

chiefly oxide, sesquioxide, or phosphate of iron. Hydrated oxide of iron (rouge) is used as a pigment and as a polishing material by glass-workers. It gives rise to a brick-red pigmentation of the lung, the other iron-compounds tending rather to blacken it.

The deposition of stone-dust, especially of quartz, flint, and glass, has been called **chalicosis** (χαλιξ grit); dust from clay, such as is inhaled by potters and makers of artificial ultramarine, gives rise to **aluminosis**.

Grinders of knives and edged tools inhale mixtures of steel-dust and grit, which cause the affection known as **grinders' asthma** or grinders' rot.

The kinds of dust just described when in small quantities give rise to no serious change in the lung other than pigmentation. This is especially true of coal-dust, of which very considerable quantities may be inhaled without perceptible injury. Metallic dust and grit are more dangerous.

Insoluble dust which is at all considerable in amount, and which cannot be got rid of from the respiratory parenchyma, sets up in addition to the above-mentioned inflammatory lesions proliferous changes in the pulmonary tissue; and these in the course of time lead to the formation in the lung of fibrous nodes and nodules. Stone-dust is the most potent factor in the production of these changes, and consequently after long-continued inhalation of gritty particles the lung becomes studded all over with numerous darkly-pigmented fibrous patches (Fig. 423 *c d*). The individual nodules vary in size from that of a millet-seed to that of a pea or cherry-stone, and when fully developed consist of layers of fibrous tissue arranged in a concentric manner (Fig. 422), with the inhaled dust enclosed between the layers. The larger nodes seem to be made up of several smaller nodules (*a*).

When the nodules are few in number (Fig. 423 *d*) and of small size, the intervening tissue is usually unaltered except as regards pigmentation, and it contains air. When the nodules are thickly studded over the lung, the tissue between them is in general condensed and indurated (Fig. 422 *e* and Fig. 423 *c* c_1), and in certain places devoid of air. Cases indeed occur in which large segments of the lung are so beset with nodules and so altered by diffuse induration of the intervening tissue that they become dense, shrunken, and airless (Fig. 423 *e*). The condition is thus appropriately described as **nodose cirrhosis** or fibroid induration of the lung. The process by which the condition is brought about is essentially a chronic indurative bronchopneumonia, inasmuch as some at least of the nodes represent simply obliterated and indurated lobules. Other nodules are produced in the lymphatics of the parenchyma or of the peribronchial and circumvascular spaces. The pleura over the cirrhotic regions is thickened and adherent (Fig. 423 *e f*). Usually the most advanced change is seen at the apices, although sometimes other portions of the lung are the chief seats of the affection (Fig. 423 *c*). In lungs of which large portions have thus become functionless, the parts that are still pervious to air often undergo compensatory emphysematous dilatation (Fig. 423 *g h*).

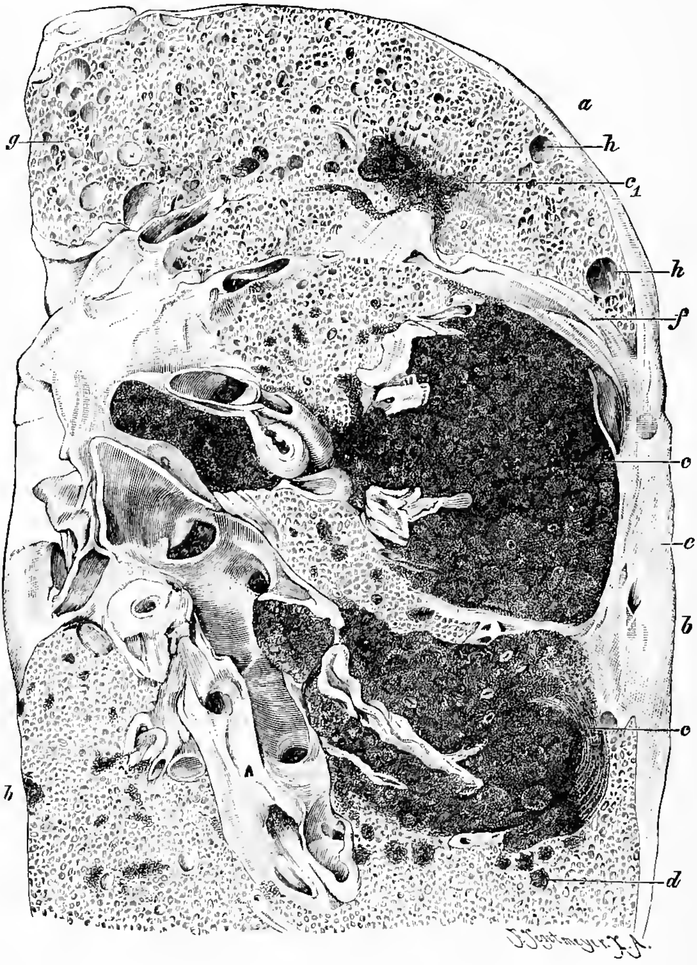

FIG. 423. CHALICOSIS OF THE LUNG.

(*Transverse* [*frontal*] *section through the posterior portion of the upper lobe and the upper half of the lower lobe of the lung: natural size*)

a	upper lobe
b	lower lobe
c	large patch of pigmented fibroid tissue made up of smaller rounded nodules
c_1	smaller fibroid patch
d	isolated pigmented fibrous nodules
e	thickened pleura
f	adherent pleurae between the lobes
g	emphysematous apex of the lung
h	emphysematous outer margin of the upper lobe

The lymphatics of the lung take their rise in the tissue-spaces of the interalveolar septa. The lymphatic radicles and vessels then run in the peribronchial, circumvascular, interlobular, subpleural, and pleural fibrous tissue. The bronchi and blood-vessels are everywhere surrounded by a close plexus of these lymphatic vessels.

Throughout the entire lymphatic system of the lungs rounded or elongated clusters of lymphoid cells are normally intercalated at various points (FRIEDLÄNDER, ARNOLD, KÖLLIKER). In children these lymphadenoid nodules are chiefly cellular, while in adults they are often more or less fibrous and pigmented. The pigment is contained in round, spindle-shaped, or stellate cells, or lies free between them.

References on Pneumonoconiosis (see also Arts. 256 and 259).

Arlidge: Diseases of occupations *B. M. J.* I **1889**

Arnold: *Staubinhalation und Staubmetastase* Leipzig **1886**; The fate of inhaled metallic dust *Ziegler's Beiträge* VIII **1890**

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Carrieu: Anthracotic pneumonoconiosis *A. de physiol.* II **1888**

Fabine: Inhalation of defibrinated blood *Cent. f. med. Wiss.* **1885**

Fleiner: Resorption of corpuscular elements by the lung and pleura *V. A.* 112 **1888**

Hanau: *Z. f. klin. Med.* XII **1887**

Harris: *Journ. of Anat.* XV **1881**

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Hirt: *Staubinhalationskrankheiten* Breslau **1871**

von Ins: Inhalation of stone-dust *A. f. exp. Path.* V **1876**

Knauff: The pigment of the respiratory organs *V. A.* 39 **1867**

Langguth: Siderosis pulmonum *D. A. f. klin. Med.* LV **1895**

Merkel: Diseases of special trades *Ziemssen's Cyclop.* I and VIII New York **1878**; *Die Staubinhalations-Krankheiten, Handb. d. Hygiene* II Leipzig **1882**

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Philip: Stone-mason's phthisis *Edinburgh Med. Journ.* XXXVII **1892**

Ruppert: Experimental researches on inhalation of coal-dust *V. A.* 72 **1878**

Sehrwald: The percutaneous injection of liquids into the trachea etc. *Gesamm. klin. Arbeiten von Rossbach u. Sehrwald* Jena **1890**

Seltmann: Anthracosis in miners *D. A. f. klin. Med.* II **1866**

Slavjansky: Experimental researches on pneumonoconiosis *V. A.* 48 **1869**

Tchistowitsch: Phagocytosis in the lungs *Ann. de l'Inst. Pasteur* III **1889**

Traube: *Deutsche Klinik* **1860**, and *Med. Times and Gazette* I **1861**; Coal-particles in the lungs *Berl. klin. Woch.* **1866**

Virchow: Black pigmentation of the lungs *V. A.* 1 **1847**, 24 **1862**, and 35 **1866**; *Edinburgh Med. Journ.* IV **1858**

Weichselbaum: *Cent. f. med. Wiss.* **1882**, and *Wien. med. Jahrbücher* **1883**

Wyssokowitsch: *Ueber die Passirbarkeit der Lunge für Bakterien* Wiesbaden **1889**

Zenker: Affections of the lung due to the inhalation of dust *D. A. f. klin. Med.* II **1866** and XXII **1878**

259. When by way of experiment animals are made to breathe for some hours air charged with decomposing and therefore irritating organic matters by means of a steam spray-apparatus, and the experiment is repeated on several successive days, the lungs are afterwards found to be studded with a multitude of minute greyish-red or yellowish inflammatory patches (Figs. 424 and 425). These disseminated lesions are due to the local

action of the irritant which has penetrated by way of the bronchioles to the respiratory parenchyma. The affection thus caused bears a close relation to the lesions of the lung produced by the inhalation of dry dust, differing from these only in the greater amount of inflammatory exudation that is poured out, and in the concomitant accumulation within the alveoli of large numbers of cells, some of them enclosing inhaled particles (Fig. 425). The lesion is accordingly described as **acute miliary bronchopneumonia** due to inhalation. It involves the infundibula and alveoli (Fig. 424), the alveolar ducts, the respiratory bronchioles (Fig. 425), and the adjacent pulmonary parenchyma.

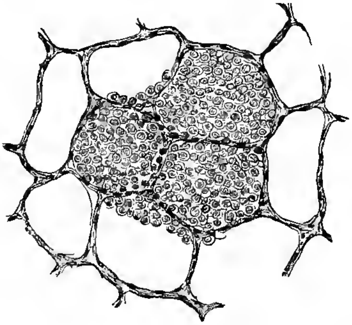

FIG. 424. MILIARY BRONCHOPNEUMONIA.

(*The inflammation extends over three alveoli: section taken from the lung of a dog after inhalation of an irritating spray:* × 80)

In man it is only under very exceptional conditions that the inhalation of irritant particles can give rise to wide-spread or general bronchopneumonia of this kind; but it is not uncommon for irritant dust to lodge in one or more segments of the lung, and therein to induce local or disseminated inflammation. Such bronchopneumonic lesions either soon recover, or more probably give place to minute patches of fibrous hyperplasia.

Larger nodose or even lobular bronchopneumonic foci are occasionally produced by the aspiration into the terminal alveoli of irritant matters derived from the mouth, nose, pharynx, larynx air-tubes, and bronchi themselves. Deleterious particles from the mouth and pharynx are drawn into the air-passages chiefly in the case of very young or comatose patients, particularly if they are subject to attacks of vomiting. In morbid conditions of the larynx and trachea associated with suppuration and necrosis, decomposing matters containing septic micro-organisms and pus may be aspirated into the lung.

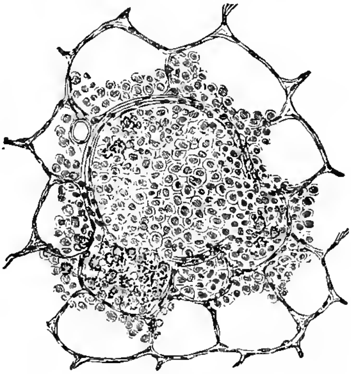

FIG. 425. MILIARY BRONCHOPNEUMONIA.

(*The inflammation extends from a respiratory bronchiole to the adjacent alveoli: some of the extravasated cells contain particles of dust: section taken from the same lung as in Fig.* 424: × 80)

The action of these substances when drawn into the lung depends naturally upon their chemico-physical characters. Large particles are apt to remain in one or other of the bronchi, and set up inflammation and obstruction of the tube. The smallest particles, when they reach the alveoli, excite more or less extensive inflammation, which may be haemorrhagic or gangrenous, croupous, catarrhal, or even purulent in character. The naked-eye appearance of the cut surface of such a lung varies with the nature of the inflammation and the stage at which it is examined; the inflamed patches may thus look grey, greyish-red, or dark-red in colour, and be uniform or granular in texture. When the exudation abounds in cells and is uncoagulated, pressure on the lung causes a turbid liquid to exude from the cut surface. This liquid varies in colour from red or greyish-red to yellowish-white, according to the relative proportions of red blood-corpuscles and of leucocytes it contains. Croupous or fibrinous exudations give the surface of section a rough and granular appearance.

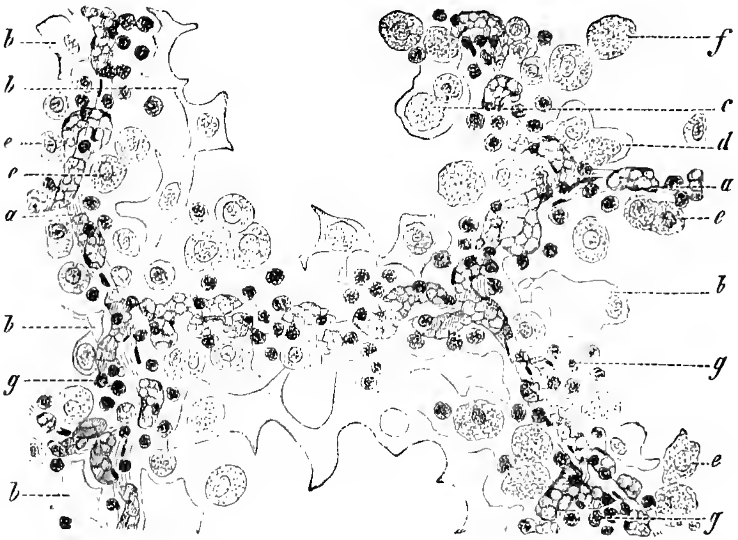

FIG. 426. SECTION OF A RECENT BRONCHOPNEUMONIC PATCH, DUE TO ASPIRATION OF LIQUID FROM THE MOUTH.

(Preparation hardened in Müller's fluid, stained with picro-carmine, and mounted in glycerine: × 200)

a interalveolar septa with distended capillaries
b desquamated epithelium, nucleated and denucleated
c epithelial plates enclosing granules and globules
d epithelial plates with granules and globules surrounding the nuclei
e small desquamated epithelial cells with visible nuclei and nearly normal protoplasm
f swollen and granular epithelial cells with indistinct nuclei
g leucocytes

Microscopic investigation shows that congestive hyperaemia (Fig. 426 *a*) supervenes very rapidly on the initial irritation. This hyperaemia is followed by serous or haemorrhagic exudation, and presently by the free extravasation of leucocytes from the capillaries, which gives the exudation the character of pus: in some cases however the exudation contains fibrin and tends to coagulate.

As soon as the exudation begins the epithelium (Fig. 426 *b c d f*) generally becomes loosened and detached. The epithelial plates (*b*) are often detached unaltered when the exudation is abundant and sudden, and are sometimes separated in the form of coherent flakes. Oil-globules however are frequently seen within them (*c*), usually aggregated around the nucleus, if the plate be possessed of one (*d*). Some of the small nucleated epithelial cells are also detached: many are scarcely altered (*e*), appearing only a little swollen; others are studded with fat-granules and globules (*f*) which often completely obscure the nucleus.

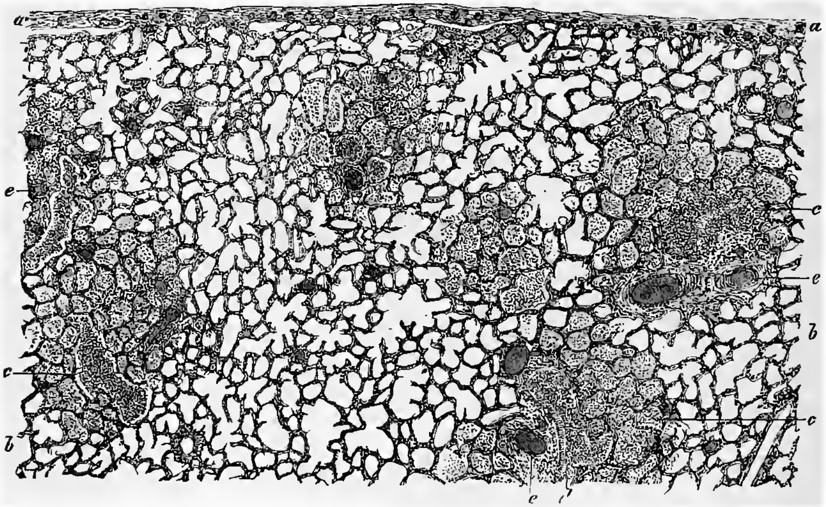

FIG. 427. BRONCHOPNEUMONIA DUE TO THE ASPIRATION OF LIQUID AND SOLID MATTERS FROM THE MOUTH.

(*Preparation hardened in Müller's fluid, embedded in celloidin, stained with haematoxylin and carmine, and mounted in Canada balsam:* × 8)

a	pleura
b	lung-tissue
c	inflammatory patch
d	longitudinal section of a bronchiole
e	transverse section of an arteriole

Mingled with the epithelial cells at an early stage are leucocytes (*g*), and in haemorrhagic inflammations red blood-corpuscles also. In cases of croupous or fibrinous inflammation, in which the exudation coagulates, threads of fibrin of various thicknesses appear between the cells, and adhere to the alveolar walls. After a time the lung is in this way studded with bronchopneumonic patches, each extending over a certain number of alveoli, and often showing in the centre an alveolar duct or a bronchiole (*d*) filled with exudation. The alveoli of the affected patch are airless and distended with exudation of one kind or another. The surrounding lung-tissue is usually hyperaemic.

Many experiments have been made on the action of saliva, decomposing organic substances, and liquids containing bacteria when aspirated into the lung. The numerous experiments on the so-called **vagus-pneumonia** are of this nature. This form of inflammation is observed after paralysis or section of the vagus and recurrent laryngeal nerves, and is due to the fact that the paralysed larynx permits saliva and foreign matters from the mouth to reach the trachea. Other investigators have conveyed into the bronchi liquids or pulverulent matters (dry or suspended in water), others again have caused animals to breathe various substances suspended in the air by means of a spray, for different periods of time.

The result of such inhalation-experiments depends on the nature of the matters inhaled and on the mode of experimentation. When finely-divided irritant substances, such as spray of sputum or of putrid liquids, are inhaled, small miliary bronchopneumonic foci are produced. When the inhaled matters are of larger bulk, we have large usually lobular patches of inflammation, haemorrhagic, suppurative, or gangrenous, as the case may be. When the foreign matters are bulky enough to occlude one or more of the bronchioles, the first effect is partial collapse or atelectasis. Specific affections of a chronic and progressive kind may be produced by the inhalation of specific micro-organisms, such as the tubercle-bacillus, which find in the lung a suitable soil for their growth and multiplication.

Large quantities of liquid quickly introduced into the lung may lead, as in **drowning**, to death by asphyxia. The liquid is carried with the inspired air into the bronchi and alveoli, and fills them with a mass of froth (PALTAUF).

References on the Inhalation of Irritant Substances (see also Art. 258).

BODDAERT: *Rech. expér. sur les lésions pulm. consécut. à la section des nerfs pneumogastriques* Brussels **1862**
BUHL, TAPPEINER, LIPPL, and SCHWENINGER: *Tagebl. der Naturforscherversammlung* Munich **1877**
COX: Experimental researches on the morbid anatomy of pulmonary inflammation *Ziegler's Beiträge* v **1889**
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FREY: *Die path. Lungenveränderungen nach Lähmung der Nervi vagi* Leipzig **1877**
FRIEDLÄNDER: *Unters. über Lungenentzündung* Berlin **1873**, and *V. A.* 68 **1876**
HEIDENHAIN: Bronchopneumonia due to hot steam *V. A.* 70 **1877**
PALTAUF: *Ueber den Tod durch Ertrinken* Vienna **1888**
SCHOTTELIUS: The action of inhaled substances *V. A.* 73 **1878**
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TAPPEINER: Experimental tuberculosis produced by inhalation *V. A.* 74 **1878** and 82 **1880**
VERAGUTH: Changes in the epithelium of the lung in experimental pneumonia *V. A.* 82 **1880**
ZIEGLER: Tuberculosis and phthisis *Volkmann's klin. Vorträge* no. 151 **1878**

260. Inflammatory processes may extend from the bronchi and bronchioles to the pulmonary parenchyma in various ways. Inflammation of the bronchial wall, characterised by hyperaemia and cellular infiltration (Fig. 428 *d*), may extend directly to the peribronchial tissue (*e*), and then by further extension invade the adjacent interlobular septa (*f*), and so induce inflammatory exudation into the alveoli themselves (*g h i*). **Peribronchitis** and **peribronchial bronchopneumonia** are thus consecutive to the initial bronchitis. In other instances bronchopneumonia follows on ordinary bronchitis, owing to the direct aspiration into the alveoli of some of the morbid contents of the bronchi. In other cases of bronchitis and bronchiolitis again some of the smaller bronchi are generally obstructed (Fig. 428 *a b*), and this gives

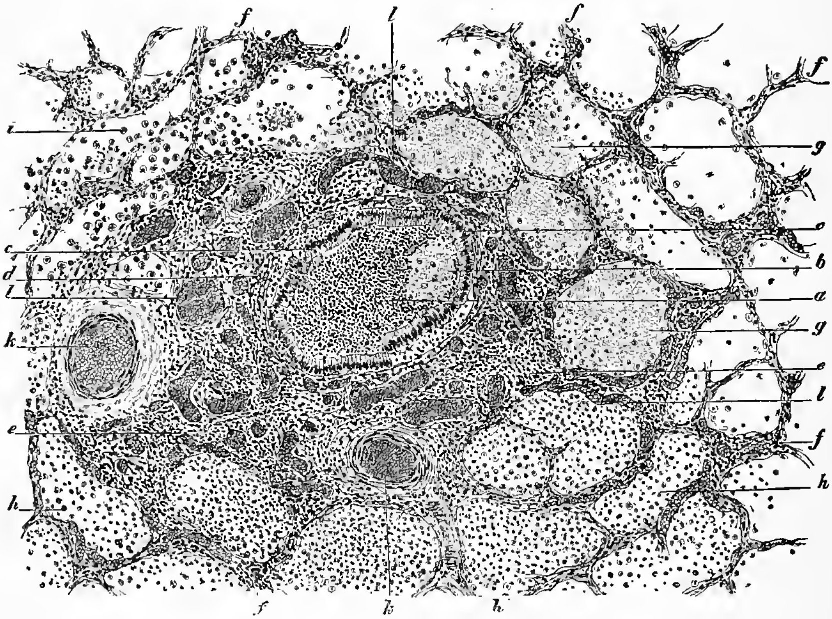

FIG. 428. PURULENT BRONCHITIS, PERIBRONCHITIS, AND PERIBRONCHIAL BRONCHOPNEUMONIA.

(*Section from the lung of a child aged fifteen months: hardened in Müller's fluid, embedded in celloidin, stained with haematoxylin and eosin, and mounted in Canada balsam:* × 45)

a	purulent bronchial contents
b	mucous bronchial contents
c c_1	bronchial epithelium beset with round-cells and partly desquamated
d	bronchial wall infiltrated with cells, its blood-vessels being distended
e	infiltrated peribronchial and periarterial connective tissue
f	septa between the pulmonary alveoli, partly infiltrated with cells
g	fibrinous exudation in the alveoli
h	alveoli filled with leucocytes
i	alveoli filled with exudation containing few leucocytes
k	transverse section of pulmonary arteries
l	bronchial, peribronchial, and interlobular arterioles distended with blood

rise to collapse or atelectasis of the corresponding lobules. When as often happens such lobules become inflamed, the extension of the inflammation may take place either by aspiration (the obstructed bronchioles becoming temporarily pervious again), or by direct spreading from the contiguous structures to the parenchyma.

The collapsed lobules assume a dark-red or livid tint, and when they are superficial the pleura over them appears to be somewhat retracted. The lungs of patients who were suffering from bronchiolitis and lobular collapse at the time of death have a marbled or mottled appearance, produced by the livid atelectatic portions in alternation with the lighter-coloured air-containing lobules. The like mottling is also apparent on the cut surface, but it is less distinct than on the pleural aspect of the lung.

But little blood escapes on pressure from the bluish-red collapsed lobules; but if they are already inflamed the livid tint gives place to a greyish, greyish-red, or greyish-yellow colour, and then a turbid liquid of corresponding appearance can be squeezed from the cut surface. This surface usually seems smooth and uniform but is in some cases granular.

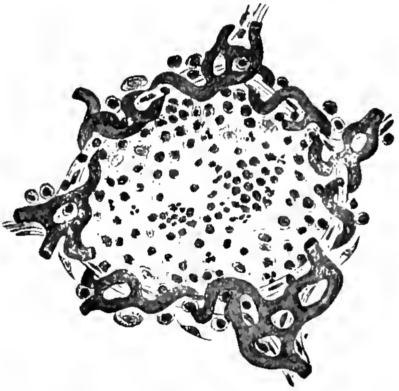

FIG. 429. CATARRHAL BRONCHOPNEUMONIA.

(An alveolus filled with liquid and large and small leucocytes: injected preparation, stained with hæmatoxylin: × 80)

The exudation is thus in general of a catarrhal nature, and the affection is accordingly described as **catarrhal bronchopneumonia,** or simply as **catarrhal pneumonia.** The alveoli contain an exudation consisting mainly of liquid (Fig. 428 *h i* and Fig. 429), with desquamated epithelium and pus-cells. Not infrequently however the exudation here and there assumes a haemorrhagic character, and consists principally of blood. Croupous exudations also, containing fibrinous coagula (Fig. 428 *g*), are sometimes met with, and in such cases the cut surface of the lung presents an appearance more or less closely resembling that of pneumonic granulation, owing to the coagulated plugs that project from the divided alveoli.

The exudation is at first always local and partial, being confined to isolated groups of alveoli, but it very frequently extends thence to entire lobules, and so gives rise to what is called **lobular bronchopneumonia.** By the coalescence of many or all of the lobular foci of inflammation within a single lobe, we may even have what is described as **lobar bronchopneumonia.**

Bronchopneumonia very frequently supervenes towards the end of life in parts of the lung that become the seat of passive hyperaemia and haemorrhagic oedema from engorgement. This form of inflammation, usually referred to as **hypostatic pneumonia**, is indicated by the turbidity of the blood-stained contents of the alveoli, and sometimes also by the granular appearance of the cut surface.

The causation of the variety of bronchopneumonia that follows upon bronchiolitis is very diverse in different cases. In the first place, according to von Besser, various pathogenic micro-organisms are apt to be present in the air-passages, for example, *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*, and *Diplococcus pneumoniae*, together with numerous non-pathogenic kinds. These organisms may remain in the air-passages without giving rise to any demonstrable lesion, but it is beyond doubt that under favourable conditions they are capable of setting up inflammation not only in the bronchial tubes, but in the respiratory parenchyma also; and hence such bacteria are commonly met with in the inflamed lung-tissue.

In the second place, the forms of bronchopneumonia that occur in the course of the different infective diseases constitute a large group; the affection being particularly common in connexion with diphtheria, measles, whooping-cough, influenza, and so on. In other words, the bronchial inflammation frequently associated with these diseases in many cases extends to the bronchioles and the alveoli. Some of these bronchopneumonic affections are due to the action of the specific virus of the primary disease; but very often they are of the nature of secondary infections, for which the primary disease has prepared the way. Thus the pneumonic affections frequently associated with influenza are referable chiefly to the dissemination and multiplication of *Diplococcus pneumoniae*, and in rarer instances of *Streptococcus* and *Staphylococcus pyogenes* (Weichselbaum, Ribbert, and others). The evidence available at present seems to show that the bronchopneumonia so commonly met with in children is in most cases due to *Diplococcus pneumoniae*, both when it appears as a complication of some infective disease and when it arises independently. The bronchopneumonia associated with diphtheria seems frequently to be due to the presence of streptococci.

Bronchopneumonia may also be caused by *Bacillus anthracis* (see the volume on General Pathological Anatomy).

References on the Morbid Anatomy and Aetiology of Bronchopneumonia (see also Arts. 258, 259, and 262).

Afanasieff: Aetiology and bacteriology of whooping-cough *Petersburger med. Woch.* **1887** (abstract in *Cent. f. Bakteriologie* III **1888**)

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EPPINGER: *Die Hadernkrankheit* (rag-dust disease) Jena **1894**

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JÜRGENSEN: *Art.* Catarrhal pneumonia *Ziemssen's Cyclop.* v New York **1877**

KROMAYER: Catarrhal pneumonia following measles and whooping-cough *V. A.* 117 **1889**

MESTER: The morbid anatomy and bacteriology of influenza *Cent. f. allg. Path.* I **1890**

MOSNY: Erysipelatous bronchopneumonia *A. de méd. exp.* II **1890**; *Étude sur la bronchopneumonie* Paris **1891**

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NEUMANN: Bacteriological investigations on the aetiology of pneumonia in childhood *Jahrb. f. Kinderheilk.* XXX **1889** (abstract in *Cent. f. Bakteriologie* VII **1890**)

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QUEISSNER: Aetiology and morbid anatomy of infantile pneumonia *Jahrb. f. Kinderheilk.* XXX **1889**

RIBBERT: Anatomical and bacteriological observations on influenza *D. med. Woch.* **1890**

WEICHSELBAUM: Bacteriology and morbid anatomy of influenza and its complications *Wien. klin. Woch.* **1890**

261. The varieties of bronchopneumonia associated with and consecutive to bronchiolitis, or caused by the occasional inhalation of irritant matters, usually terminate in resorption of the exudation and recovery. The exfoliated epithelium is made good by the regenerative proliferation of the remaining epithelial cells. There are however other forms which are accompanied by such severe and irreparable changes that, if death does not intervene, they leave permanent lesions behind them.

In most forms of bronchopneumonia the peribronchial and interalveolar tissues are often infiltrated with cells to such an extent that it is difficult to distinguish the pulmonary parenchyma from the alveolar contents. When the inflammation is due to pyogenic micrococci, circumscribed **abscesses** are formed here and there in the lung, each surrounded by a zone of infiltrated and often blood-stained and granular-looking tissue. In other cases the pulmonary tissue becomes necrotic, the process then assuming the character of gangrenous bronchopneumonia, the discoloured and putrid patches of **gangrene** being surrounded by tissue infiltrated with blood and sometimes with fibrin or pus. This form of bronchopneumonia is generally consecutive to putrid bronchitis set up by the aspiration into the lung of putrid or septic matter, or to anthrax.

These processes, if they are not immediately fatal, may end in recovery by the removal of the necrotic tissue and the absorption or expectoration of the exudation. The loss of substance is made good chiefly by the compensatory expansion of adjacent parts,

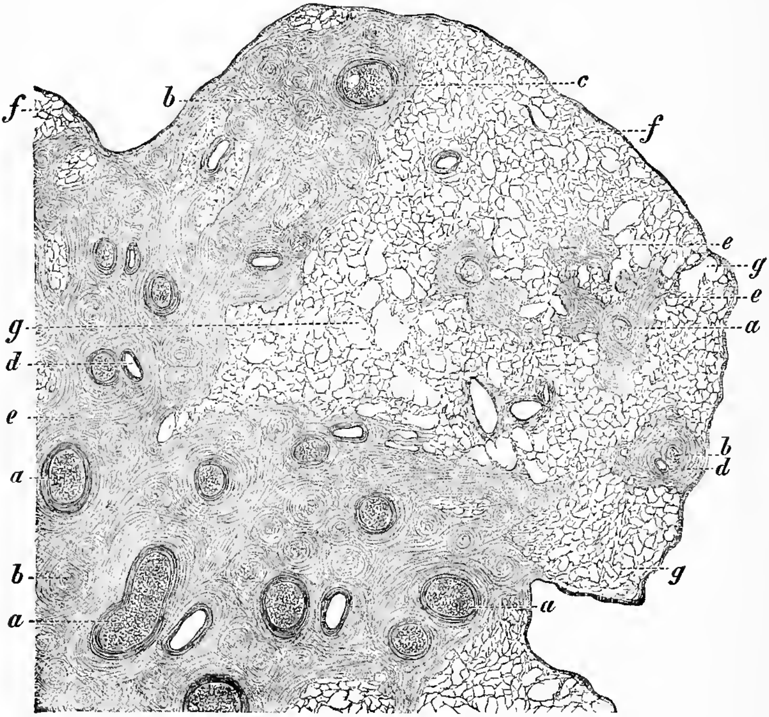

FIG. 430. INDURATION OF THE LUNG FOLLOWING BRONCHOPNEUMONIA AND BRONCHIAL OBSTRUCTION.

(Horizontal section through the consolidated apex, stained with picro-carmine, and mounted in Canada balsam: × 5)

a larger bronchi plugged with inspissated secretion
b obliterated bronchioles
c small bronchus distended with secretion
d pulmonary arterioles
e indurated and contracted pulmonary tissue
f normal pulmonary tissue
g emphysematous pulmonary tissue

but there always is also a certain amount of **induration** of the lung-tissue due to fibrous hyperplasia about the local seat of the inflammatory process. Such fibroid induration is occasionally associated with non-necrotic inflammations, especially those accompanied by chronic obstruction of the bronchial tubes (Fig. 430 *a*) with permanent collapse of a segment of the lung.

The apex is the most frequent seat of induration and contraction of this kind (Fig. 430 *e*): indeed, apart from tuberculosis, apical consolidation is probably oftenest due to prolonged or recurrent attacks of bronchopneumonia, induced it may be by the inhalation of irritating dust or soot. In the latter case the indurated lung is always mottled or uniformly stained with black, and the condition is therefore known as **grey cirrhosis** or slaty induration.

Induration of the lung may however also follow upon acute infective bronchopneumonia, and so is occasionally observed in children in the form of fibrous cicatrices, some of which may actually enclose residues of necrotic tissue.

The formation of new fibrous tissue which gives rise to these indurations takes place partly in the interalveolar septa and peribronchial and interlobular tissues (Fig. 420 *a c*), and partly within the alveoli, the granulations that spring up in the latter situation being supplied with blood-vessels from the alveolar walls. Proliferation of the epithelium also takes place in some cases, and where the alveoli are impervious to air a structure not unlike that of the foetal lung may be thereby produced, inasmuch as the alveoli become lined with cubical epithelial cells.

When a bronchopneumonic patch lies immediately beneath the pleura that membrane is involved in the inflammatory process, and is overlaid with a fibrinous or fibrino-purulent deposit. When recovery ensues **pleural thickenings** and adhesions are usually left behind.

Suppurative and gangrenous inflammations of the lung in new-born infants are usually bronchopneumonic, and due to aspiration of the liquor amnii or of septic genital secretions. Metastatic inflammations arising from infection of the umbilical stump are also met with. The pleura and the interlobular fibrous tissue are in general gravely affected by the inflammation.

262. **Lobar croupous pneumonia** is an infective disease of the lung, usually due to *Diplococcus pneumoniae*, but capable of being produced by other micro-organisms, such as in particular *Bacillus pneumoniae*, and *Streptococcus* and *Staphylococcus pyogenes*. Certain other bacteria also have the power of inducing croupous inflammation extending over an entire lobe of the lung.

In most cases we cannot make out with certainty by what channel the micro-organisms that give rise to croupous pneumonia have gained access to the lung, or in what manner they multiply within it. We know only that during the course of the affection the microbes in question are found in the inflammatory exudation, usually also in the sputum and in the pulmonary tissue, and occasionally in other organs as well; but we are unable to demonstrate in what way they have reached the lung. In a few special cases the histological appearances are such as to suggest that the microbes have entered the lung-tissue by way of the bronchi, in others they appear to reach it by way of the circulation.

As a rule croupous pneumonia is an infective disease of which the pulmonary lesion is the only or at least the most striking and important local manifestation. In some instances however it makes its appearance in the course of a disorder that is either general or localised in other organs, such for example as acute articular rheumatism, typhoid fever, influenza, malarial fever, erysipelas, septic osteomyelitis, meningitis, nephritis, or the like: in this case the pneumonia is probably to be regarded as a secondary local manifestation of the primary disorder, having in some degree the characters of a metastasis. It is however to be specially noted that lobar pneumonia arising in the course of specific infective diseases, such as typhoid fever or influenza, is usually due to the same kind of microbes as give rise to ordinary primary lobar pneumonia. Even in cases in which the consolidated lung contains the typhoid bacillus, the presence of pyogenic micrococci or pneumococci can usually be demonstrated also.

Lobar croupous pneumonia is distinguished by the presence, in the alveoli, the bronchi, and the lymph-vessels, of a fibrinous exudation which when recent consists of desquamated epithelium, leucocytes, red blood-corpuscles, and reticular filaments of fibrin (Fig. 431). It should not however be forgotten that the composition of this exudation varies greatly in different cases, both at the outset and during the several stages of the disease.

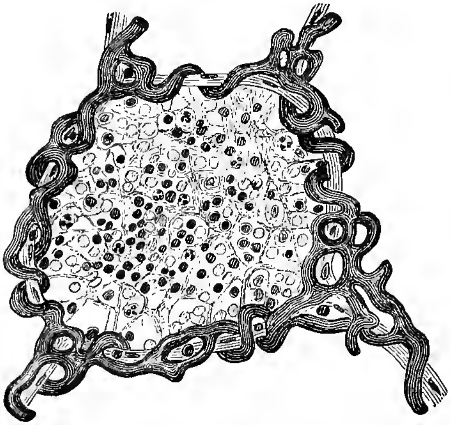

FIG. 431. PULMONARY ALVEOLUS FILLED WITH FIBRINOUS EXUDATION.

(*The exudation consists of desquamated epithelium, leucocytes, red blood-corpuscles, and fibrinous filaments: preparation injected from the pulmonary artery with Prussian blue, hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 80)

The exudation sometimes abounds in cells, the fibrin, which is usually deposited in the form of fine granular filaments, being scarcely discernible; in other cases the cells are scanty or in some alveoli all but absent, and then the fibrinous mesh-work is the predominant feature. The filaments are sometimes very slender and with no clear outline, looking like mere rows of granules; sometimes they are stout, lustrous, and sharply-defined. The proportion of red blood-corpuscles present may be large or small, and within the same lung often varies in different alveoli. When the exudation does not coagulate, but remains as a turbid liquid containing a varying proportion of cells, which can be squeezed out of the lung by pressure, the inflammatory process assumes the characters of lobar catarrhal pneumonia (Art. 254).

The process of exudation into the alveoli begins with congestive hyperaemia, the affected segment of the lung becoming deep-red in colour, and abnormally firm in consistence. This is the stage of **congestion.** When the exudation coagulates the lung becomes almost as solid as the liver, and this stage is accordingly described as that of **hepatisation.** If at the time of examination the cut surface is red from congestion and from admixture of blood with the exudation, the condition is called **red hepatisation.** If on the other hand the lung on section is greyish or yellowish-grey in colour, owing to emptying of the blood-vessels and decolorisation of the exudation, the condition is spoken of as **grey** (greyish-yellow or yellow) **hepatisation.** Grey hepatisation is the commonest variety seen *post mortem*, because patients who succumb to the disease usually die several days after the process has begun ; but different parts of the same lobe often exhibit different tints. It is moreover possible that the red-tinted portions are not always those most recently affected, for hyperaemia and indeed haemorrhagic extravasation are liable to supervene even in the stage of resolution.

A hepatised portion of the lung is firm and airless, and a varying quantity of turbid liquid can be expressed from its cut surface; this liquid comes most freely during the stage of resolution. When the lung has been compressed, the surface of section presents a granular appearance (pneumonic granulation), owing to the projection of the fibrinous contents of the alveoli above the general level. The pleura over the affected parts of the lung is always inflamed, and looks turbid from the presence of a delicate fibrinous film, or is overlaid with a thick yellowish false-membrane. The costal surface of the swollen and consolidated lung is often marked with shallow impressions of the ribs.

The extent of the consolidation differs widely in different cases: sometimes it spreads over the greater part or the whole of one lung, sometimes it involves only a single lobe or a part of one. In certain cases parts of two contiguous lobes are hepatised, and the characteristic appearances are occasionally found in a number of isolated patches, though within each the infiltration is in general uniformly diffused (Fig. 432), and there is no special centre of exudation. Sometimes however the consolidated patches contain yellowish nodular foci, which may be regarded as centres of infection, and minute examination reveals the existence of a few excavations in the tissue, corresponding to bronchioles and alveolar ducts, whose contents include an exceptional number of cells and but a small proportion of fibrin. The lung-tissue in the neighbourhood of the hepatised portions may be hyperaemic, anaemic, or oedematous. The peribronchial lymph-glands look swollen and sodden, and are red, reddish-grey, or grey in colour. The bronchi are also inflamed, and contain a mucous or muco-serous secretion which is tinged red or reddish-brown by the extravasation of red blood-corpuscles, and in the later stages of the disease is mingled with the liquefied fibrinous contents of the bronchioles and alveolar ducts. Sometimes croupous casts are formed in the smaller non-respiratory bronchi.

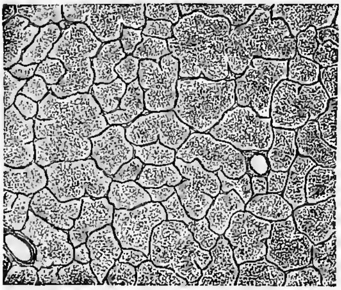

FIG. 432. CROUPOUS HEPATISATION OF THE LUNG.

(*Preparation hardened in Müller's fluid, and stained with alum-carmine:* × 20)

During the course of an attack of pneumonia inflammation makes its appearance in other organs, such as the pericardium, meninges, and mediastinal tissue, the mucosa and submucosa of the pharynx, soft palate, and nasal cavities, the conjunctiva, kidneys, etc., in which case the affected structures can be shown to contain micrococci.

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263. The structure of the lung is always maintained unimpaired throughout an attack of croupous pneumonia which runs an ordinary course, and its textural characters are accordingly recognisable at all stages of the disease (Fig. 432). Experiments show that the vascular system also is uninjured, and can be injected in the usual way. Accordingly in most cases croupous pneumonia ends in **recovery,** the exudation being re-absorbed and in part expectorated.

Re-absorption and expectoration of the fibrinous exudation are rendered possible by the **liquefaction** of the latter. This begins with the loosening of the fibrinous adhesions to the alveolar walls, followed by the disintegration of the coagulum itself. In the lung of a patient dying from seven to ten days after the onset of the disease, most of the alveolar coagula will be found to have become loosened from the walls. In cases of death during the second to the fourth week the alveolar contents are seen to be in various stages of disintegration and resolution, though some of the alveoli may contain fibrinous residues, and where the inflammation has been intense fresh coagula may have formed in other alveoli. In cases that run a favourable course the exudation is more rapidly re-absorbed, and clinical observation shows that many croupous consolidations of the lung disappear in a very short time indeed.

During the stage of **resolution** the contents of the alveoli include at first a large proportion of cells, especially in the outer parts of the consolidated region. The accumulation of cells in these parts is due partly to a fresh extravasation of leucocytes, partly to desquamation of proliferous alveolar epithelium, in other words to secondary catarrhal inflammation consecutive to the fibrinous exudation. The cells lying in the alveoli show signs of fatty degeneration, cloudy swelling, liquefaction, and disintegration. The pulmonary tissue itself is somewhat infiltrated with cells. By degrees the proportion of cells in the alveolar contents diminishes, as they are removed by re-absorption and to some extent by expectoration. The process of resolution and recovery is completed by the re-entrance of air into the bronchioles and alveoli, the regeneration of the desquamated pulmonary epithelium, and the complete removal of the exudation.

The pleuritic exudation is re-absorbed at the same time, some proliferation with consequent thickening and adhesion of the serous surfaces usually taking place in the membrane.

Destructive textural changes resulting from pneumonic consolidation of the lung are on the whole of rare occurrence, though cases are met with in which the infiltrated tissue here and there undergoes suppuration or gangrene. In **suppuration** of the lung the tissue gradually becomes yellowish-white and breaks down by colliquation, while pus-corpuscles collect within it in large numbers. In **gangrene,** on the other hand, the tissue assumes a dirty-grey tint, and is converted into a tindery or pulpy slough with an intensely foetid odour. When a gangrenous patch is subpleural, the overlying serous membrane is sometimes raised in blebs or bullae. Both suppuration and gangrene are probably in most cases attributable to some special form of infection, the primary pneumonia itself being due to some unusual cause, such as pyogenic micrococci, or to a mixture of several different kinds of microbes; or the subsequent changes are brought about by a second invasion of a new kind. It sometimes happens however that the suppurating lung-tissue contains none but the ordinary pneumococci (ZENKER). The co-existence with the pneumonia of foetid bronchitis and bronchiectasis undoubtedly favours the supervention of septic infection resulting in gangrene of the lung. Croupous pneumonia in a lung previously sound and healthy, and not infected with the tuberculous virus, never issues in caseation.

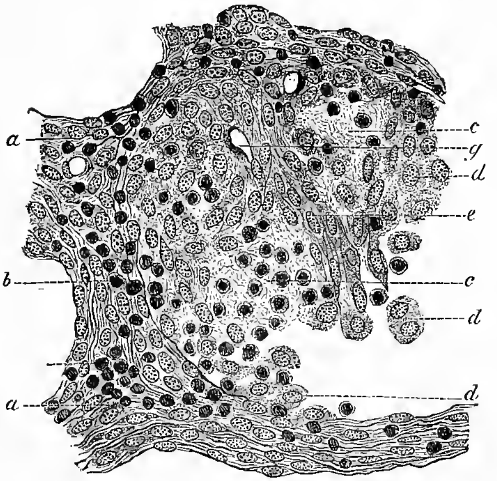

FIG. 433. INTRASEPTAL AND INTRA-ALVEOLAR GROWTH OF FIBROUS TISSUE.

(*Stained with haematoxylin:* × 150)

a	alveolar wall thickened and fibroid
b	alveolar wall thickened and infiltrated with small round-cells
c	cellular exudation within the alveolus
d	intra-alveolar formative cells (fibroblasts)
e	string of fusiform fibroblasts
g	newly-formed intra-alveolar blood-vessel

It is however by no means rare for cases of pneumonia to terminate in more or less extensive pulmonary induration or **cirrhosis.** The ultimate causes of this result cannot in general be determined: it may be due to a special kind of infection, or to some pre-existing peculiarity in the condition of the lung.

The processes that issue in fibroid induration consequent upon croupous pneumonia can sometimes be observed in active development in patients dying from four to ten weeks after the first onset of the disease. In such cases the lung is in the very peculiar condition appropriately described as **carnification**: it is red, flesh-like, somewhat firm, and tough. At the first glance the lung looks as if it were in a state of red hepatisation, but the organ is less rigid, and lacks the characteristic pneumonic granulation. The vessels of the carnified tissue are full of blood, as the colour indicates, and the alveoli sometimes contain extravasated red blood-corpuscles. The tissue is moreover densely infiltrated with cells, which are sometimes so numerous that they obscure the textural details of the pulmonary structure. The essential point indeed consists in the presence both within the alveoli and in the tissue itself of a multitude of cells, derived for the most part from proliferous fibrous tissue and epithelium. The pulmonary tissues are in fact in a state of active hyperplasia, associated with more or less abundant extravasation of leucocytes from the vessels. In some of the alveoli remains of the antecedent fibrinous exudation can still be discovered.

The hyperplastic proliferation of the pulmonary structures results in the production of new fibrous tissue, leading to thickening of the interalveolar septa (Fig. 433 *a*), and to the development of vascular granulation-tissue (*d e g*) within the alveoli. Sometimes certain of the fibrinous coagula occupying the alveolar cavities are seen to be pervaded by vascular fibrous tissue, others are largely or wholly replaced by it, and some alveoli are entirely filled by masses of granulation-tissue. The bronchial, peribronchial, circumvascular, interlobular, and pleural connective tissue are likewise hyperplastic, with the ultimate result that the lung itself becomes condensed and indurated and the pleural surfaces coherent.

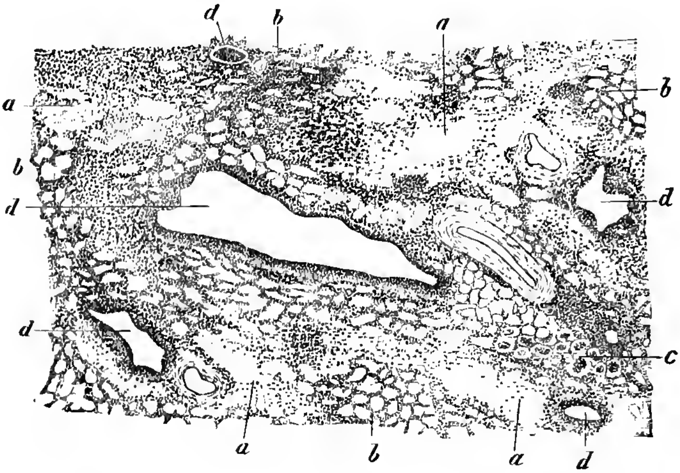

FIG. 434. CIRRHOSIS OF LUNG CONSECUTIVE TO CROUPOUS PNEUMONIA.

(*Preparation hardened in alcohol, and stained with carmine:* × 15)

a dense pigmented fibrous tissue
b alveoli with thickened and infiltrated septa
c alveoli filled with cells
d dilated bronchi infiltrated with cells

The precise course followed by the pulmonary induration depends as regards its details upon various circumstances. If the alveolar epithelium has been destroyed by the antecedent affection, the proliferous septa may become coherent and thicken into continuous tracts of dense fibrous tissue (Fig. 434 *a*). The like result is produced when the alveolar cavities become filled up with vascular granulation-tissue whencesoever arising. Simultaneous proliferation of the septal connective tissue and of the alveolar epithelium gives rise to new fibrous tissue that is highly cellular, and encloses gland-like loculi lined with cubical epithelium. If air should regain admission into this compact tissue, it may once more become vesicular, its loculi or vesicles being bounded by thickened walls (*b*).

The extent of the induration that thus results from an attack of pneumonia varies very greatly. It may be limited to the stratum immediately underlying the pleura, or extend over the greater part of a lobe. It may be continuous, that is, uninterrupted by islands of air-containing tissue, or it may take the form of fibrous bands traversing the parenchyma in various directions and not very sharply marked off from it. This kind of cirrhosis is always indeed distinguishable by the fact that the fibrous tissue takes the form not of sharply-defined nodes or nodular clusters, but of strands and patches that pass gradually into the air-containing tissue. This characteristic feature is absent or ill-defined only when secondary bronchopneumonia or peribronchitis complicates the simple cirrhotic process.

The pleura overlying the cirrhotic patches is usually thickened and adherent to the costal pleura. The patches give rise to shrinking and puckering of the surface, the intervening alveoli being emphysematous. After a time, if the induration and contraction are at all extensive, the corresponding bronchi are distorted and more or less dilated (Fig. 434 *d*); sometimes they are also ulcerated. For months and it may be years a chronic inflammation of the bronchi and of the pulmonary parenchyma is thus kept up in the indurated region.

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264. **Haematogenous septic pneumonia** is usually a secondary lesion, due to metastasis from a focus of suppurative or gangrenous inflammation in some other part of the body. In some cases it is the principal manifestation of a pyaemia whose primary origin is undetected (cryptogenetic septic pyaemia). The metastatic forms commonly start in some infected external wound or some internal suppuration, such as a perityphlitic or parametritic abscess, purulent meningitis, abscess of the brain or liver, suppurative osteomyelitis and arthritis, dysentery, strangulated hernia, right-sided endocarditis, etc.

The character of the morbid processes initiated by the metastasis depends on the nature of the matter conveyed to the lung by the blood. If its particles are of some bulk they may lodge in and block the arteries; if it consists of finely-divided particles they may reach the capillaries and thence set up inflammation in the parenchyma.

When infective matters enter the circulation from a septic wound, some may be arrested in the lung and give rise to **embolic infarction**. Suppurative inflammation is set up around the infarcted tissue, by means of which the latter becomes encircled by a zone of yellowish infiltration, and presently is loosened and separated from the tissue about it. The infarct then naturally undergoes necrosis and breaks up under the action of continued suppuration, so that at length there is formed a cavity filled with pus, a **metastatic abscess** of the lung. If the septic embolus contain putrefactive organisms, or if these enter the infarct from the bronchi, the tissue may undergo putrid decomposition or **gangrene**, and so be transformed into a foul dirty-grey or slate-coloured slough.

When the original irritant reaches the vessels of the lung in the form of very fine particles, such as micrococci, which are not arrested till they reach the capillaries and there lodge, the resulting patches of inflammation are usually small and ill-defined. At first the inflammation is often haemorrhagic in character, but no infarct is formed, and ultimately the affected parts either recover or suppurate and become gangrenous.

When suppuration begins, yellowish spots appear in the red or greyish-red infiltrated tissue, and these gradually break down into small abscesses. As gangrene supervenes the lung-tissue assumes a dirty-brown tint, and softens into a foul-smelling pulp.

In recent cases microscopical examination shows that the inflammatory exudations are in part haemorrhagic and in part cellular or fibrinous, and the blood-vessels very often contain microbes in process of multiplication. When the infection is due to the pyogenic staphylococci, many of the capillary blood-vessels are seen to be crammed with these organisms.

Should the septic embolism be subpleural, the pleura is always simultaneously inflamed. The pleuritic exudation is purulent or fibrino-purulent, and sometimes extends over the entire surface of the lung.

Suppuration and gangrene within the lung occasionally extend by continuity to the neighbouring structures. The inflammation thus set up is usually haemorrhagic and fibrinous in character, and speedily passes into suppuration and gangrene. Soon the process reaches the peribronchial and interlobular lymphatics, and they become filled with serous, fibrinous, and purulent exudations, while the tissue about them becomes infiltrated with cells. This lymphangitis and perilymphangitis may start either from an embolic abscess of the lung or from a purulent pleurisy. In the latter case the interlobular tissues are the most affected.

The embolic abscesses may break through either into the bronchi or into the pleural cavity, the former being the commoner event. When adhesions unite the lung to the thoracic wall or to the diaphragm, rupture may take place to the exterior or into the abdomen.

The smaller abscesses generally heal up more or less perfectly by absorption of the pus, the larger by rupture or evacuation: granulations are formed around the abscess cavity, and subsequently are transformed into cicatricial tissue. If the absorption of the pus is incomplete it sometimes becomes inspissated and calcified. Adhesions between the apposed serous surfaces are invariably the result of the healing of the pleuritic patches.

Metastatic pneumonia taking the form of suppurative or gangrenous inflammation is probably in all cases due to infection by the ordinary pyogenic micrococci. The circumscribed or patchy form of pneumonia, other than that originating in the bronchi, which arises in the course of specific infective diseases such as typhoid fever, anthrax, small-pox, and so on, may however be caused by the specific virus of the primary disease.

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265. When the pleura is inflamed (pleurisy) the underlying pulmonary tissue is liable to become involved also, owing either to simultaneous injury or infection of the latter, or to secondary extension of the inflammatory process from the serous membrane to the lung: the condition thus induced is appropriately described as **pleurogenous pneumonia** (Fig. 435).

The extension of the inflammation from the pleura to the lungs takes place chiefly by way of the lymphatics (*c*) that pass from the interlobular septa of the pleura (*a*) to the peribronchial tissue, so that the process assumes the characters of interlobular, circumvascular, and it may be even peribronchial **lymphangitis** (*c*).

The interlobular and peribronchial lymphatics become inflamed in connexion with various forms of pleurisy; but such lymphangitis is particularly frequent in the case of certain purulent and fibrino-purulent inflammations of the pleura that are of a pyaemic nature, and due either to metastasis from some suppuration within the lung or to some primary infection of which the pleurisy is the only local manifestation. Children appear to be especially predisposed to inflammation of the interlobular connective tissue; it is at all events observed more frequently in children (Fig. 435) than in adults. The affection is not uncommon in infants suffering from pyaemia due to septic infection of the umbilical stump.

The distension of the lymphatics with purulent or fibrino-purulent exudation (*c*) causes the lobules to appear as if separated by bands of yellowish-white infiltration, and the veins (*g*) to be surrounded by similar zones. The peribronchial lymphatics are often affected in like manner, each bronchus on section appearing to be surrounded by a sharply-defined collection of pus, and usually containing a purulent secretion in its lumen. If the interlobular fibrous tissue itself undergoes suppuration, some of the lobules are apt to become loosened and isolated from each other. This form of pulmonary inflammation is accordingly spoken of as **dissecting pneumonia**.

The tissue lying between the dilated lymphatics is more or less compressed, dark-red in colour, hyperaemic, and airless. The alveolar tissue generally becomes inflamed by direct extension from the pleura (*f*), as well as from the interlobular septa (*d*), and the inflammation may in the end spread over a considerable part of it (*e*). Infection and inflammation of the pulmonary parenchyma may in the same way be induced by antecedent inflammation of the peribronchial and periarterial lymphatics. The inflamed lung-tissue becomes airless, appears saturated with a serous exudation containing cells, and assumes a greyish-red or greyish-yellow colour. Here and there the exudation may be tinged with blood.

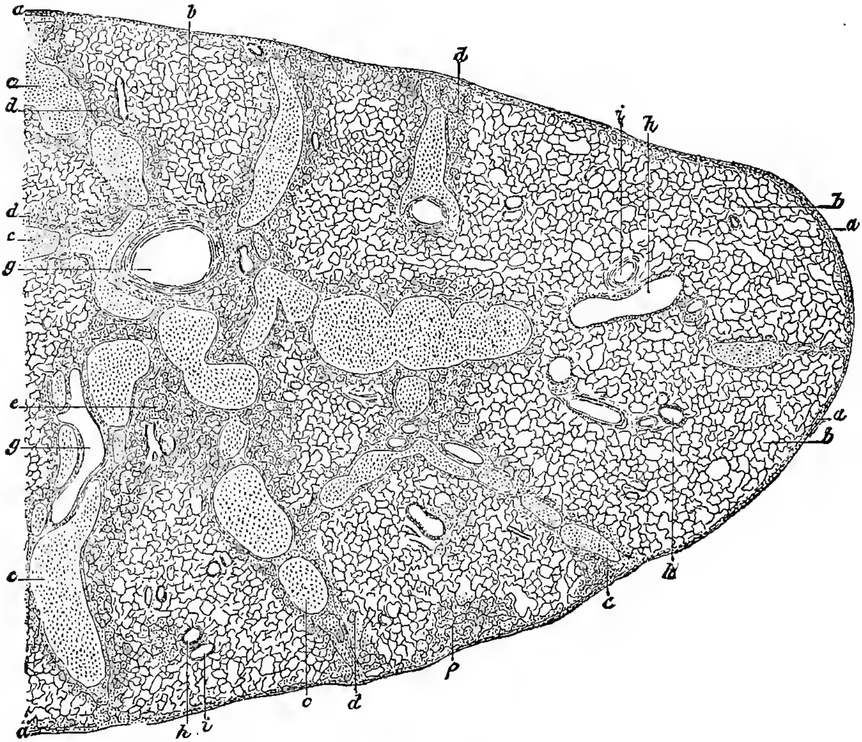


FIG. 435. INTERLOBULAR PNEUMONIA WITH LYMPHANGITIS INDUCED BY PURULENT PLEURISY.

(Section of the lung of a child aged four years: hardened in Müller's fluid, stained with picro-carmine, and mounted in Canada balsam: × 70)

a	pleura
b	normal lung-tissue
c	lymph-vessels distended with inflammatory exudation
d	pneumonic infiltration in the neighbourhood of the lymphatics
e	diffuse pneumonic infiltration
f	subpleural pneumonic patch
g	veins
h	bronchi
i	arteries

If the attack is not fatal, recovery takes place by resorption, though in most cases there remains some permanent thickening of the interlobular and peribronchial tissues.

Chronic inflammations of the pleura, associated with hyperplasia of the pleural connective tissue (Fig. 436 *a*), are also liable to extend to the interlobular septa (*c*) and peribronchial connective tissue (*e*), the lung at length appearing to be traversed by firm bands of fibrous tissue, which form a kind of coarse meshwork starting from the thickened peribronchial tissue (*e*).

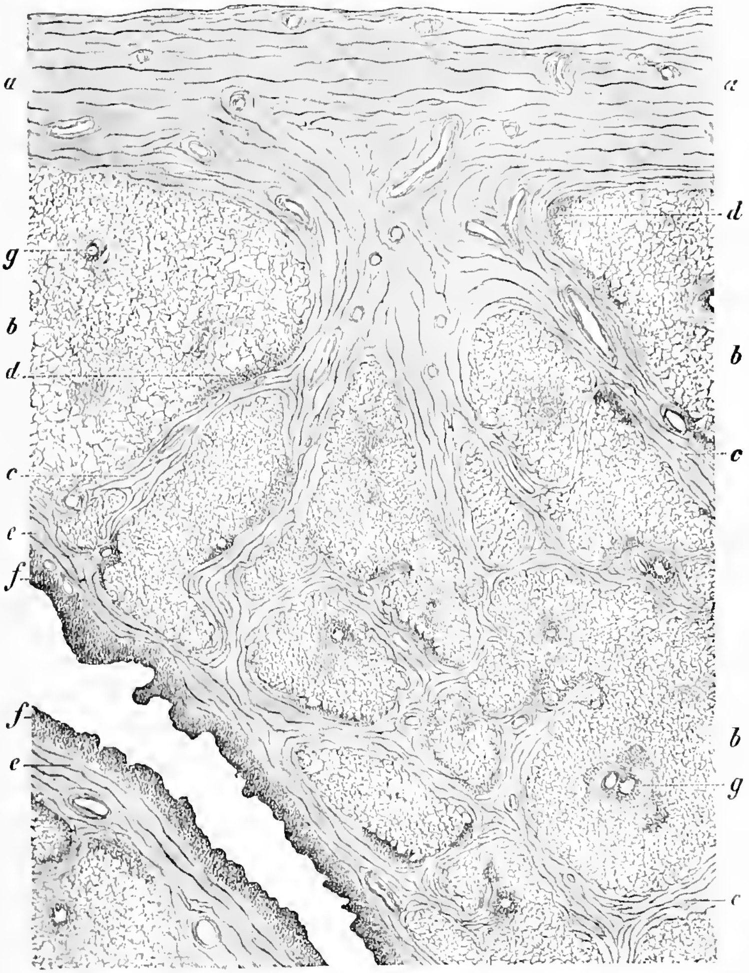


FIG. 436. CHRONIC PLEURISY AND PLEUROGENOUS INTERLOBULAR PNEUMONIA.
(*Preparation hardened in Müller's fluid, and stained with picro-carmine*: × 3·5)

a thickened pleura
b pulmonary tissue
c thickened interlobular septa
d cellular infiltration at the junction of the septa with the pulmonary alveolar tissue
e thickened peribronchial tissue
f dilated bronchus with infiltrated mucous membrane
g bronchioles with infiltrated walls

If the inflammation and fibrous hyperplasia extend further and involve the alveoli (*d*), the latter may become infiltrated and ultimately cirrhotic.

When a part of the lung is compressed by pleuritic effusion or by contraction of the thickened pleura and interlobular septa, the affected alveolar tissue generally becomes more or less collapsed and airless. By the cohesion of the walls of the collapsed alveoli the compressed portions of the lung sometimes undergo permanent consolidation and induration.

In these conditions the bronchi are usually affected by catarrhal inflammation, which results in cellular infiltration of the mucosa (*f g*). As the new fibrous tissue contracts, the bronchi are often distorted (*f*) and dilated, partly by the traction of the shrinking interlobular septa and partly by the internal air-pressure which acts on them in an abnormal manner (Art. 248).

Inflammation of the other contents of the thorax or of the abdomen sometimes extends to the lungs. The mediastinal tissues and peribronchial lymph-glands, the oesophagus, the spinal column, the stomach, and the liver are the parts most commonly concerned; and according to the character of the primary affection the resulting inflammation of the lung is purulent, gangrenous, or indurative. Thus an abscess of the liver breaking through the diaphragm may give rise to an abscess at the base of the lung.

In ulcerative disease of the lung the bronchi sometimes become perforated. An abscess at the base, for instance, or a broken-down caseous bronchial gland, may rupture into a neighbouring bronchus. If the matters thus evacuated are infective or otherwise irritating, and if some of them are aspirated into other parts of the parenchyma of the lung, secondary bronchopneumonia generally results.

Traumatic lesions of the lung, caused for example by a fractured rib, give rise in the first place to haemorrhage and perhaps to entrance of air into the pleural cavity (**pneumothorax**). If the wound is not contaminated the rent is healed by thrombosis and subsequent cicatrisation. Septic infection of the wound results in suppuration and gangrene of the lung.

CHAPTER LXXXVI

TUBERCULOSIS AND OTHER INFECTIVE GRANULOMATA OF THE LUNG

266. **Tuberculosis** of the lung may be brought about in three ways, the specific bacillus gaining access to the pulmonary tissue through the blood-vessels, through the lymphatics, or through the air-passages. We may conveniently describe the corresponding varieties as haematogenous, lymphogenous, and pneumatogenous, respectively.

Haematogenous tuberculosis is generally of the nature of a metastatic process, the bacillus entering the blood-vessels from some distant focus such as a tuberculous lymph-gland, and so being conveyed to the lung. Instances however are on record in which no such starting-point could be discovered. The affection takes the form of miliary tuberculosis or of a circumscribed metastatic lesion.

Haematogenous miliary tuberculosis is characterised by the appearance in the lung-tissue of an eruption of grey tubercles, which afterwards undergo caseous degeneration; they are either distributed uniformly over both lungs and pleurae, or are aggregated in an irregular manner or confined to particular parts of the lung. The characteristic cellular clusters appear first in the alveolar parenchyma, and the earliest and youngest tubercles are accordingly of the most diverse shapes—crescentic, annular, stellate, and so on (Fig. 437 *a*). Presently however the alveoli and minuter bronchioles surrounding each cluster become filled up with cells, and the growing cluster is thus rounded off into a solid globular nodule or miliary tubercle, with perhaps a few cellular projections and offshoots corresponding to the alveolar septa that intersect it. Within the site occupied by the tubercle the existing capillaries are obliterated, and a mature tubercle is thus invariably non-vascular.

The eruption of tubercles is accompanied by a certain degree of hyperaemia, and a lung affected with miliary tuberculosis is accordingly abnormally firm and of a dark-red colour. As a rule the affected parts still contain air, though this is less in amount than should correspond to the apparent volume of the organ. The hyperaemic bronchi frequently contain blood-stained mucus similar to that met with in croupous pneumonia.

When the miliary eruption is abundant and extends over both lungs, death usually ensues; but a second variety exists in which the eruption is scanty and circumscribed, and this variety is not necessarily fatal. The tubercles in the latter case increase in size and become caseous, and so form the starting-point for further morbid changes.

Circumscribed haematogenous tuberculosis is produced in the same way as miliary tuberculosis, but differs from it in that the eruption of tubercles is limited to one or two isolated spots in the lung. Its general course is similar to that of the second variety of miliary tuberculosis above referred to, and it gives rise to like secondary changes in the pulmonary tissue.

Lymphogenous tuberculosis, in which the bacilli are conveyed to the lung by way of the lymphatics, commonly has its origin in a tuberculous bronchial lymph-gland, or a tuberculous lesion of some of the bones enclosing the thorax, such as the vertebrae in particular. The lymph-glands themselves usually become affected owing to antecedent disease of the lung; but inhaled tubercle-bacilli sometimes gain access to the lymph-glands directly from the bronchi or even the alveoli, without producing any permanent tissue-changes in the lung on their passage through it.

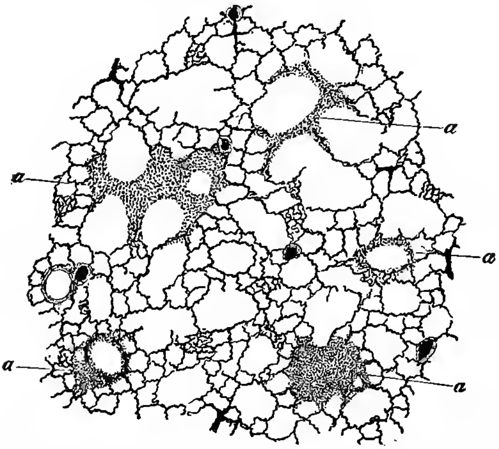


FIG. 437. MILIARY TUBERCULOSIS OF THE LUNG.
(*Preparation injected, and stained with carmine :* × 30)
a tubercles of various shapes

When the bronchial glands undergo caseous degeneration, the adjacent tissues of the lung, bronchi, or trachea are at times invaded by direct extension of the inflammatory process, and so ultimately permit the caseous matter to break through and escape into the air-passages. Moreover, a more or less wide-spread eruption of tubercles may appear in the parts of the pleura contiguous to the diseased glands, the eruption proceeding along the course of the subpleural lymphatics. In like manner secondary infection of the pleura and lung occasionally takes place in the neighbourhood of caseous foci in the bones, and gives rise to single or multiple tuberculous lesions in the lung.

Pneumatogenous tuberculosis, in which the infection is conveyed by way of the air-passages, is due to the inhalation of air containing tubercle-bacilli, or to the intrusion into the respiratory tubes of tuberculous matter from some previously-infected source, such as a tuberculous bronchial gland or laryngeal ulcer, or of liquid from the mouth containing tubercle-bacilli.

If no other irritant gains access to the lung along with the in-

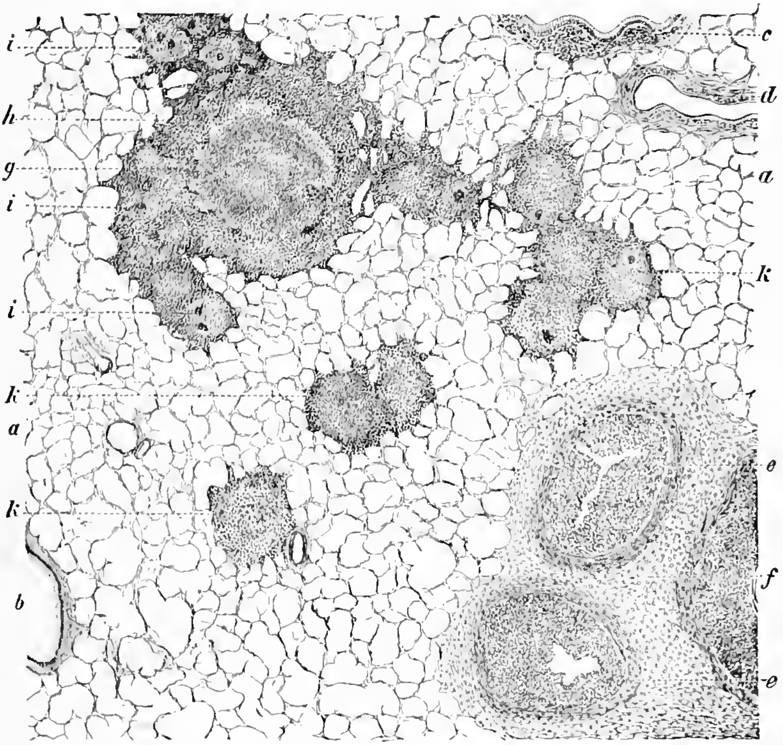


FIG. 438. PRIMARY TUBERCULOUS FOCI IN THE LUNG, WITH COMMENCING TUBERCULOUS LYMPHANGITIS.

(Section from left apex of the lung of a woman aged 25, which contained a few scattered nodes with central caseation: preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam: × 15)

a normal lung-tissue
b normal bronchus
c bronchus with inflamed and infiltrated wall
d artery
e caseous masses encapsuled in connective tissue
f fibroid induration
g caseous centre, and
h cellular periphery of a tuberculous node
i k resorption-tubercles in the adjacent lymphatics

haled tubercle-bacilli, and if these do not forthwith perish but proceed to multiply at their point of settlement, they give rise to the formation of tubercles, built up of proliferous epithelial and connective-tissue cells together with immigrant leucocytes from the blood-vessels. It is probable that the initial point of settlement and multiplication of the tubercle-bacilli is either within an alveolus or in one of the pulmonary lymphatics. Some of the bacilli in process of multiplication are taken up by the proliferous or other cells. The number of primary foci thus produced naturally depends upon the number of bacilli that have been drawn into the lung. Probably in most cases but one or two bacilli are inhaled at any one time; but when for example a tuberculous gland ruptures into a bronchus, a multitude of bacilli are thereby simultaneously introduced and diffused by aspiration over many of the air-tubes.

In adults the primary foci of pneumatogenous tuberculosis are usually situated at the apex; but in children no particular part of the lung is more likely than another to be affected. The foci sometimes, and this more especially in children, have the appearance of ill-defined patches of bronchopneumonia (Fig. 439 *a*), with a cellular exudation that presently becomes caseous; in other cases, chiefly among adults, the foci take the form of rounded fairly-definite nodes with caseous centres (Fig. 438 *g h e*). In the course of time these nodes may become more or less completely encapsuled by the proliferous growth of fibrous tissue around them (Fig. 438 *f*); they then cease to be active and sometimes long remain stationary or at length undergo calcification. It is thus no uncommon occurrence to find on post-mortem examination that the lung encloses completely-encapsuled caseous nodes with no recent tubercles in their neighbourhood.

More frequently however, when no such encapsulation takes place by the formation of fibrous tissue, the process of cellular multiplication and infiltration continues to extend from the peripheral parts of the primary node along the contiguous interalveolar septa, and the tuberculous focus thus increases in size. By and by fresh tubercles develope in the immediate vicinity of the primary node (*i*), as well as in the lymphatics (*k*), plainly showing that the tubercle-bacilli spread by way of the lymph-channels.

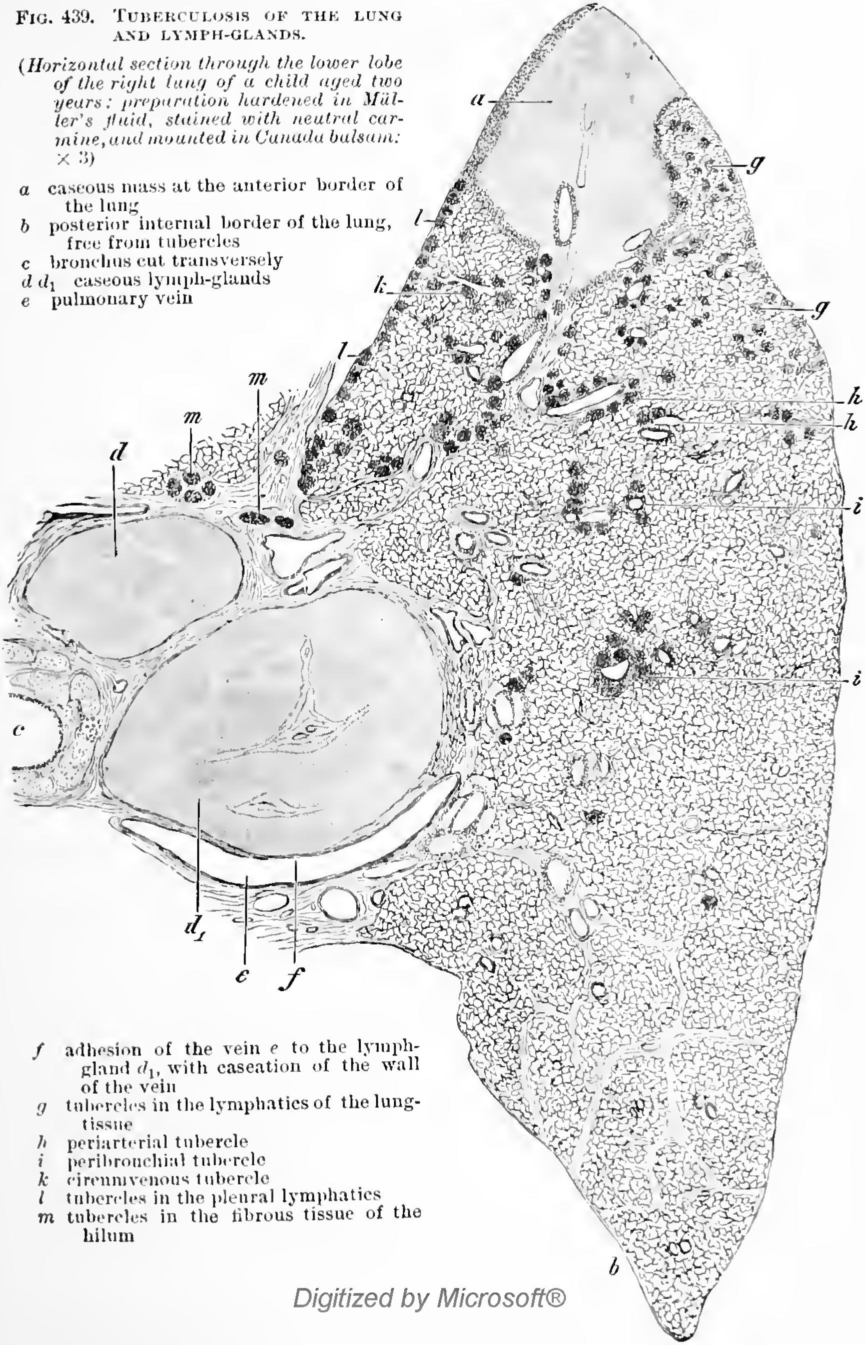
No general rule can be laid down as to the ultimate extension of such tuberculous lymphangitis. The lung is very abundantly provided with intralobular, interlobular, peribronchial, circumvascular, and subpleural lymphatics, whose trunks communicate with the peribronchial lymph-glands; and often enough tubercles make their appearance along the course of all these channels (Fig. 439 *g h i k l m*), until the affected lobe is more or less completely pervaded by clusters of lymphangitic nodes and nodules.

In children (Fig. 439) the process usually advances rapidly, the primary tuberculous focus (*a*) soon undergoing caseous degeneration, while tubercles appear along the course of the various lymph-channels. The bronchial glands (*d* d_1) are nearly always attacked at an early stage, and become caseous as soon as the primary lesion. Such caseous glands not infrequently become adherent to the neighbouring veins (*e f*), arteries, or bronchi, and

FIG. 439. TUBERCULOSIS OF THE LUNG AND LYMPH-GLANDS.

(*Horizontal section through the lower lobe of the right lung of a child aged two years: preparation hardened in Müller's fluid, stained with neutral carmine, and mounted in Canada balsam:* × 3)

a caseous mass at the anterior border of the lung
b posterior internal border of the lung, free from tubercles
c bronchus cut transversely
d d_1 caseous lymph-glands
e pulmonary vein



f adhesion of the vein *e* to the lymph-gland d_1, with caseation of the wall of the vein
g tubercles in the lymphatics of the lung-tissue
h periarterial tubercle
i peribronchial tubercle
k circumvenous tubercle
l tubercles in the pleural lymphatics
m tubercles in the fibrous tissue of the hilum

tubercles and caseous deposits are thereupon produced in their walls. A caseous bronchial or tracheal gland in contact with one of the systemic veins sometimes becomes adherent to and infects the vessel-wall in the same way.

Haematogenous tubercles occasionally increase in size in a similar manner, and lead to the formation of lymphangitic nodules.

The pulmonary tissue between the nodules usually remains for long unaltered (Fig. 438), and continues to admit air to the alveoli. In the immediate neighbourhood of the growing tubercles, however, disturbances of the circulation with inflammatory exudation and proliferation are generally induced, whereby the alveoli become airless and consolidated; and the tissue thus altered is apt in the end to undergo caseation. Induration is however a somewhat commoner result, giving rise to the formation of greyish or slate-coloured patches that enclose clusters of grey or yellowish caseous nodules or larger caseous nodes. When these patches are close together they are apt to coalesce, and thus encroach more and more on the air-containing tissue till it is reduced to a few strips and islands. In the end an entire lobe or other segment of the lung may become converted into a firm solid airless mass enclosing a certain number of caseous patches.

After a certain time the caseous portions of the lung sometimes become calcified, but more frequently they soften, and at the same time increase in extent by caseous disintegration of the tissue about them. If this tissue should happen to be already indurated the process of caseation and softening advances slowly; when it is merely infiltrated, the spread of the caseous disintegration is often rapid. In either case however the result is the formation of a closed cavity (or **vomica**) containing pulpy detritus, often including recognisable shreds of broken-down lung-tissue, or greyish-white pus-like matter.

Should the destruction of the circumjacent tissue proceed very slowly or be altogether arrested for a while, such a cavity may remain completely shut off for a long period of time, and its semiliquid contents may even become inspissated and calcareous. Usually however the cavity gradually enlarges as the tissue enclosing it again becomes studded with new tubercles, and then caseates and breaks down. If the walls of the nearest bronchus, hitherto shut off from the cavity, are themselves attacked by the destructive process, the contents of the cavity sooner or later break a way into the lumen of the open tube. In this way the caseous detritus, together with a certain number of tubercle-bacilli, enters the air-passages and mingles with the sputum, giving rise on the way to more or less intense inflammation in some part of the respiratory tract. Once this has happened it is always possible that, the bronchial mucous membrane being attacked, the tuberculous inflammation may extend by way of the bronchi to other portions of the lung, especially if the contents of the bronchi, including the bacilli, should be forcibly aspirated into the alveoli.

To judge from clinical experience, such an accident does not appear to occur frequently, and when it takes place it often leads to the infection of comparatively small portions of the lung. Instances however are on record in which such aspiration of infective matter has suddenly infected the whole of both lungs, and the virulent particles must therefore have penetrated every ramification of the bronchi. Cases of this kind have been described in which the contents of a pulmonary cavity were evacuated into a bronchial tube, or in which a caseous gland had ruptured into the trachea or one of the larger bronchi. Physical exercises such as dancing, jumping, and the like, which are liable to induce powerful inspiratory efforts, are of course likely to favour the aspiration of the tubercle-bacilli. A sudden acute outburst of pulmonary tuberculosis, which had previously been latent and inactive, has thus been known to follow violent physical exertion.

The consequence of the aspiration of infective particles is the formation of a secondary bronchopneumonic tuberculous focus (Fig. 440). Corresponding to the degree of virulence and the distribution of the aspirated matter the resulting lesion takes the form of small circumscribed cellular nodules, or of large infiltrated patches that at first are somewhat diffuse and ill-defined. The virulence of the infective matter varies in different cases, and in addition to the specific virus it may include other irritants such as micrococci; while on the other hand it is known that all individuals do not react equally to a given irritation.

When the irritation excited is moderately intense the course of the secondary bronchopneumonia is in general as follows. An exudation abounding in cells is poured out and the irritated tissue becomes proliferous; in a few days a cellular nodule containing bacilli (Fig. 440 *f h*) is thus produced, and speedily becomes caseous in the centre (*f*) while its periphery (*g*) still consists of living and active cells. As the nodule grows older this peripheral zone usually becomes fibro-cellular in character. The lung-tissue around the nodule is the seat of an exudative inflammation whose intensity naturally differs in different cases. As a rule the neighbouring alveoli (*i*) contain extravasated liquid and leucocytes, desquamated epithelium, and often fibrin (i_1). The alveolar septa are partially infiltrated with small round-cells, especially in the neighbourhood of the veins (*k*). The adjacent lymphatics, peribronchial and periarterial (*d*), as well as interalveolar and interlobular (*l*), are also in some measure affected by the inflammation, being more or less distended by the exuded matters (*d l*). When the nodule is subpleural the pleura is simultaneously inflamed.

When a number of tuberculous bronchopneumonic foci are thus formed by aspiration, each passes through much the same series of changes as were described in the case of the primary focus. Some become converted into caseous or fibro-caseous nodes and nodules, which may occupy entire lobules and lead to progressive tuberculous lymphangitis, others soften and break down and by aspiration of their contents lead possibly to fresh bronchopneumonic infection.

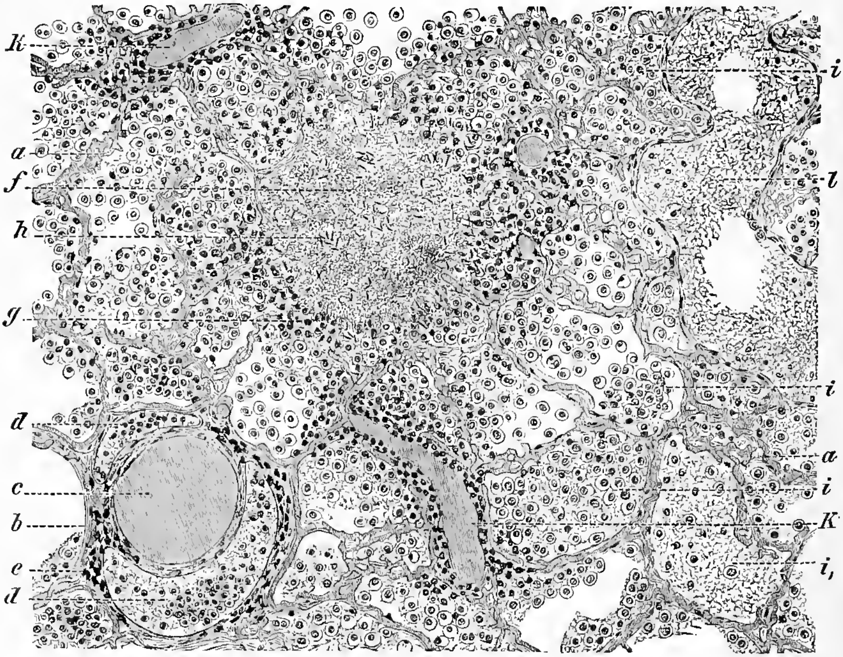


FIG. 440. MILIARY TUBERCULOUS BRONCHOPNEUMONIA.

(Secondary patch due to the aspiration of the contents of a small caseous node which ruptured into a bronchus: preparation injected with blue gelatine and stained with alum-carmine: the bacilli drawn from a parallel section stained with fuchsin: × 80)

a	interalveolar septa with injected capillaries
b	respiratory bronchiole
c	injected artery
d	circumvascular lymphatic distended with exudation
e	pigment lying round the lymphatic
f	caseous centre, and
g	fibro-cellular periphery of the bronchopneumonic patch
h	tubercle-bacilli (× 160)
i	cellular and fibro-cellular exudation in the alveoli
i_1	chiefly fibrinous exudation
k	vein with surrounding cellular infiltration
l	interlobular lymphatic distended with exudation

Partial obliteration of the capillaries always takes place at the site of the tuberculous patch, and in the caseous parts the circulation is cut off altogether. Very often the tuberculous proliferation extends to the walls of the veins or arteries, and when the diseased wall yields and disintegrates, aneurysmal dilatation and rupture, with arterial haemorrhage (**haemoptysis**), are not uncommonly the result. Should the tuberculous disintegration extend to the intima of an artery or vein without inducing rupture or thrombosis and obliteration of the vessel, tubercle-bacilli may gain access to the blood, and be carried by it to other parts of the lung or into the systemic circulation.

Tuberculous infection of the blood sometimes takes place also from caseous lymph-glands, when these become adherent to contiguous blood-vessels and induce tuberculous changes in the vessel-walls (Fig. 439 $d_1 f$).

Tuberculous affections of the lung, in which the local lesions are not numerous, sometimes undergo complete **recovery**, the lesions becoming encapsuled in fibrous tissue: in other cases their further progress is so checked that no local extension of the disease takes place for many years. So long however as any living bacilli are present, it is obvious that the recovery must from a pathological point of view be regarded as imperfect.

When the tuberculous lesions in the lung are already numerous, some of these may doubtless recover completely or partially; but it is extremely unlikely that recovery should simultaneously take place in all. So long therefore as a single tuberculous focus continues to undergo disintegration and thus to afford the tubercle-bacilli a suitable nidus for their growth, so long the risk and indeed the probability remain that the destructive process will again advance by way of the lymphatics, the blood-vessels, or the bronchi.

The walls and the contents of many tuberculous vomicae contain not only tubercle-bacilli but other micro-organisms, particularly micrococci, and occasionally mould-fungi also. Some of these are merely saprophytes, yet it is not unlikely that the products of the decompositions they induce in the necrotic detritus act injuriously on the surrounding structures. At times pathogenic microbes find a nidus in the wall of the cavity, and give rise to duplex or multiple infection of the lung.

267. In cases of death from chronic pulmonary tuberculosis (Fig. 441) the lung generally presents certain characteristic appearances. The pleural surfaces (*d*) are more or less adherent and thickened; the lung itself contains indurated and consolidated patches (*c*); cavities (*e*) exist at the apices; and tuberculous nodes and nodules (*g i k*) are scattered through other parts of the lung. Ulcers are moreover frequently present in the bronchial walls.

In adults the most advanced changes are usually found at the apices (Fig. 441), and in the majority of cases consist of slate-coloured or whitish patches of **induration** (*c*), enclosing grey, caseous, or even calcified tuberculous nodules of various sizes. The consolidated patches are due to fibrous hyperplasia of the tissue surrounding the nodules, induced by the inflammation they excite.

No general rule can be laid down as to the extent of this fibrous induration, but it frequently affects the greater part of the upper lobe, and very often involves the whole of the apical region, while the lower portions of the upper lobe and the upper parts of the lower lobes contain only circumscribed patches of induration separated by air-containing tissue.

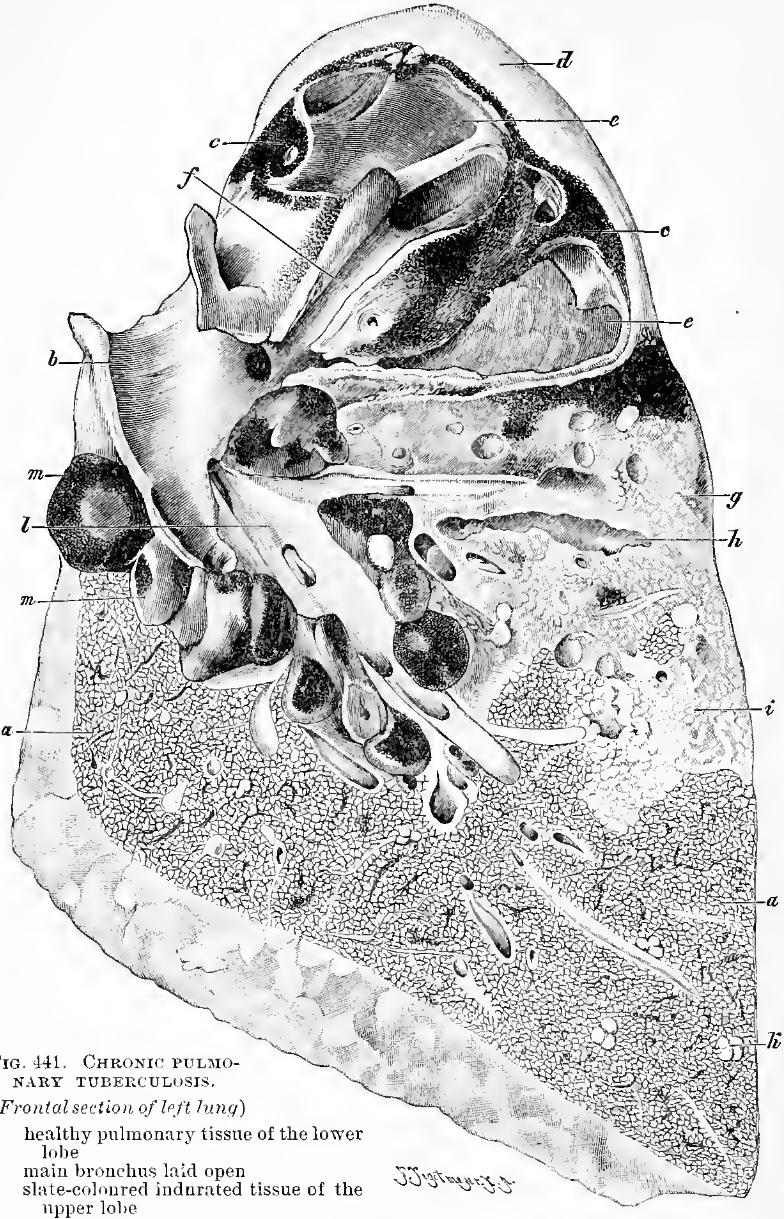


FIG. 441. CHRONIC PULMONARY TUBERCULOSIS.

(*Frontal section of left lung*)

a healthy pulmonary tissue of the lower lobe
b main bronchus laid open
c slate-coloured indurated tissue of the upper lobe
d thickened pleura
e smooth-walled cavities
f bronchus communicating with an apical cavity
g indurated pulmonary tissue with greyish-white nodules
h dilated and ulcerated bronchus
i pulmonary tissue beset with grey translucent and yellowish-white caseous tubercles
k small cluster of tubercles
l pulmonary artery
m enlarged and pigmented lymph-glands

The cavities known as **vomicae** (*e*) are usually produced by the caseous degeneration of the pulmonary tissue, their communications with the bronchi being secondary and accidental. Frequently however cylindrical, saccular, or fusiform bronchiectases (Art. 248) are formed within the indurated patches, and afterwards progressively increase in size by the gradual disintegration of their walls.

The size and number of the apical cavities are very different in different cases. Sometimes only a single vomica no larger than a hazel-nut is present; in other cases almost the entire upper lobe is excavated and converted into one large cavity; in others again the upper half of the lobe is riddled with excavations more or less completely separated off from one another.

The cavities contain air and yellowish-white or greyish-white caseous pus, mingled with necrotic detritus and sometimes with liquid or coagulated blood. The walls of these cavities are sometimes ragged and uneven, sometimes smooth and indurated (*e*) or lined with caseous granulations; they appear as if smeared with caseous pus. Projecting ridges and bands of pulmonary tissue traversing its interior often subdivide a cavity into a series of sinuses or of more or less freely intercommunicating loculi.

The remnants of tissue within the cavities usually consist of the bronchi, arteries, and veins, surrounded with indurated fibrous tissue. When haemorrhage has taken place into the cavity an eroded or ruptured vessel, often already occluded by thrombosis, can generally be found in the wall of the cavity or in one of the bands that traverse it. In some cases the rupture of such a vessel is preceded by aneurysmal dilatation and protrusion of its inner through a rent in its outer coat.

The end of a bronchus (Fig. 441 *f*) where it enters a cavity usually appears as if it were cut off abruptly; sometimes however the bronchial wall seems to run for a short distance in the wall of the cavity. Tuberculous ulcers are frequently found in the walls of the bronchus near the cavity, or even at some distance from it.

The lower lobes (Fig. 441 *a*) usually contain air and are reddened; they enclose a varying number of pale-grey or whitish nodules (Fig. 441 *k* and Fig. 442) often clustered in groups of three or four like a clover-leaf, and surrounded by a zone of tissue that is hyperaemic, infiltrated, and greyish-red, or indurated and slate-coloured.

On close examination it will be seen that these nodules are for the most part small bronchopneumonic patches, within which the pulmonary tissue is broken down (Fig. 442 *a b c d*) and therefore incapable of injection (Fig. 443 *a*). They are probably due to the aspiration into the bronchioles (Fig. 442 *e* and Fig. 443 *b*) or alveolar ducts and alveoli (Fig. 442 *b c d* and Fig. 443 *a c*) of tubercle-bacilli, which have given rise to inflammation and proliferation *in situ*. Accordingly the patches are usually adjacent to or entirely surround one of the branches of the pulmonary artery (Fig. 442 *a b c* and Fig. 443 *a b*). The individual patches consist chiefly of a caseous and disintegrated central portion (Fig. 443 *a b c*) and a fibro-cellular peripheral zone sometimes enclosing giant-cells. From the point of view of its mode of origin and structure the condition might fitly be described as a **fibro-caseous nodular bronchopneumonia**.

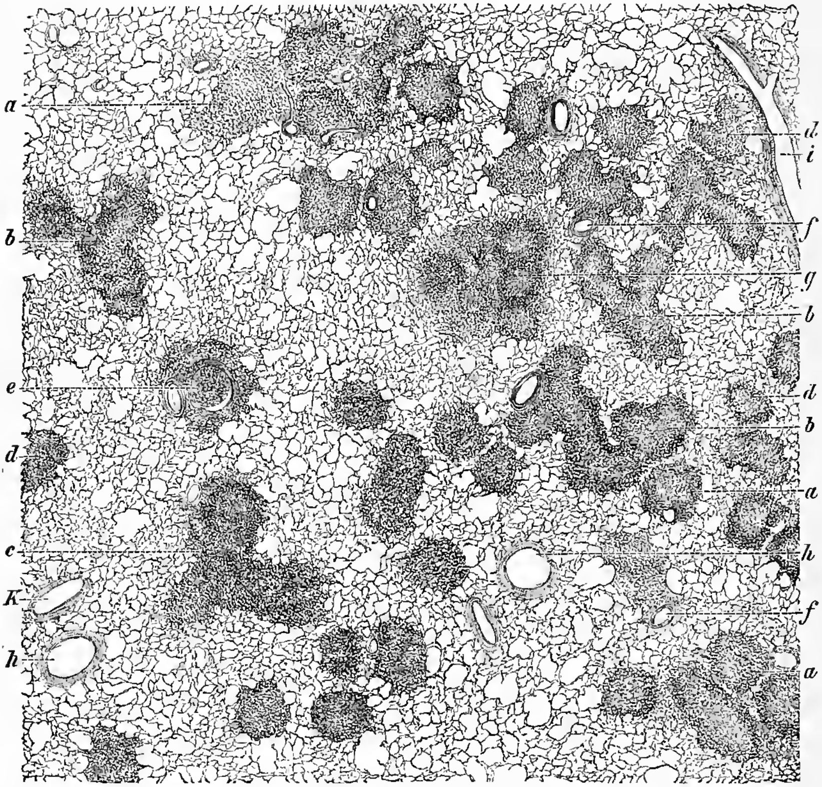


FIG. 442. CHRONIC NODULAR TUBERCULOUS BRONCHOPNEUMONIA.
(*Preparation hardened in Müller's fluid, and stained with picro-carmine*: × 6)

a b c d	tuberculous nodes of various forms corresponding to systems of alveolar ducts	*f*	arteriole
e	section through an infiltrated and occluded bronchiole	*g*	groups of nodules in process of coalescence
		h	small bronchus (normal)
		k	artery

As the fibro-caseous nodules increase in number and size, and encroach on the intervening functional air-containing tissue, the greater portion of the lung passes into a condition of nodose induration and generally of contraction also, which in many respects resembles the cirrhosis produced by the inhalation of dust; it is therefore described as **nodose tuberculous cirrhosis**, or fibroid phthisis (Fig. 444). The visceral layer of the pleura is usually much thickened (Fig. 444 *a*) and adherent to its costal layer.

This form of chronic tuberculosis usually extends over both lungs, but is more advanced in one lung than the other: cases have indeed been noted in which one lung was almost or entirely unaffected. Far-advanced and extensive fibro-caseous tuberculous cirrhosis is of course possible in one lung only, as otherwise the amount of available respiratory tissue would be insufficient to maintain life. When one part of the lung is rendered functionless by the disease the remainder becomes hypertrophic and emphysematous by way of compensation.

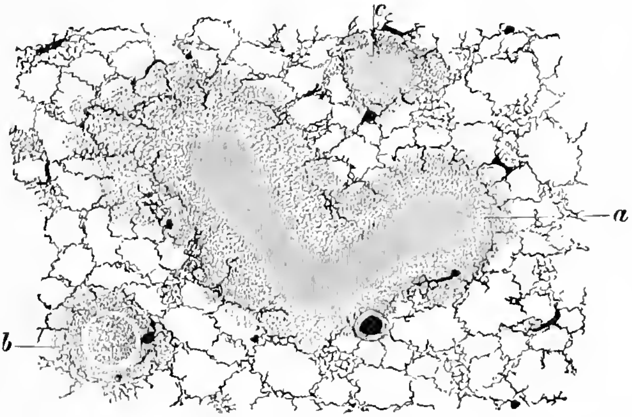


FIG. 443. NODOSE TUBERCULOUS BRONCHOPNEUMONIA.

(*Preparation injected with Prussian blue from the pulmonary artery, and stained with carmine:* × 25)

a V-shaped patch, caseous in the centre and fibro-cellular at the periphery, produced by the infiltration of two contiguous alveolar ducts and their alveoli

b respiratory bronchiole with cellular exudation in and around it

c alveolar duct with caseous contents and infiltrated alveoli

268. The variety of chronic pulmonary tuberculosis described in Art. 267 is as a rule not rapidly fatal, and may last for many months or even years: sometimes indeed it progresses very slowly or comes to a complete standstill. But it too often happens that a case which originally appeared chronic and unprogressive, with a tendency to cirrhotic induration, suddenly becomes acute and virulent. In other cases the process assumes a virulent character from the outset. Thus a small cavity, that has perhaps given rise to no appreciable symptoms and is enclosed in a small patch of indurated tissue, may suddenly become the starting-point of a rapid dissemination of tuberculosis throughout the ramifications

of the bronchi. Cases occur in which within a very short time the lung becomes studded with innumerable minute grey and white bronchopneumonic patches, due to bronchial aspiration, and having much the appearance of a miliary haematogenous eruption. Having regard to the way in which this variety is produced we might describe it as **pneumatogenous miliary tuberculosis,** or miliary disseminated tuberculous bronchopneumonia.

In other cases the disease is virulent from the outset, inasmuch as the tuberculous patches speedily become caseous and softened, with little tendency to the formation of firm fibrous tissue. Rapid disintegration of the affected parts is the natural result, cavities are soon produced, and the metastatic foci that are formed tend in like manner to undergo caseous degeneration. The outcome is that in a very short space of time the lung is studded with caseous patches and riddled with cavities. The process might thus be termed **caseative** or **ulcerative caseous tuberculosis**; clinically it is sometimes spoken of as *phthisis florida.* From its pathogenesis it might be called **nodular caseous tuberculous bronchopneumonia.** The softening and disintegration of the pulmonary tissue are sometimes so extreme that the process almost resembles rapid suppuration.

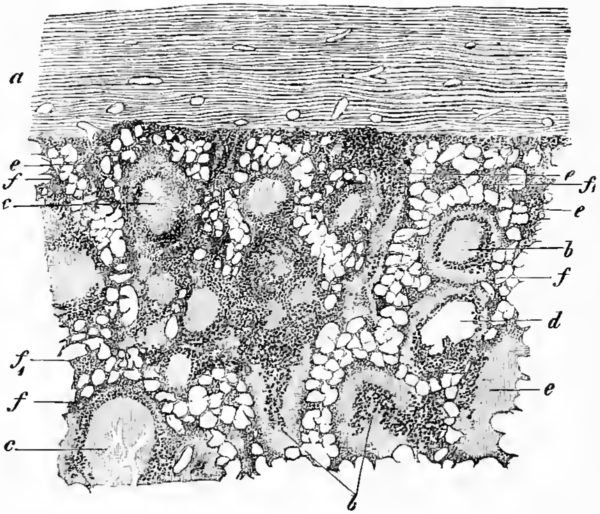


FIG. 444. CHRONIC NODOSE TUBERCULOUS CIRRHOSIS.

(Preparation hardened in alcohol, and stained with haematoxylin: × 20)

The persistent alveolar septa are in parts infiltrated with cells, and the thickened and indurated tissue is pigmented.

a thickened and fibroid pleura
b fibro-caseous indurated nodes
c bronchioles with caseous contents and thickened walls
d small bronchiectatic cavities
e thickened interlobular septa
f recent cellular infiltration surrounding the indurated nodules and (f_1) involving the lymphatics and their neighbourhood

In this form of tuberculosis the separate patches are usually larger than they are in the indurative form, and they readily coalesce to form still larger patches. Not uncommonly the infiltration spreads about the lesions over entire lobules or groups of contiguous lobules, which thus become at first greyish-red, then undergo grey hepatisation, and finally turn yellowish, opaque, and caseous. In this way the nodular variety passes into **caseous lobular bronchopneumonia** (Fig. 445). By the coalescence of many such infiltrated lobules an entire lobe may become caseous, and the result is occasionally described as **caseous lobar pneumonia.**

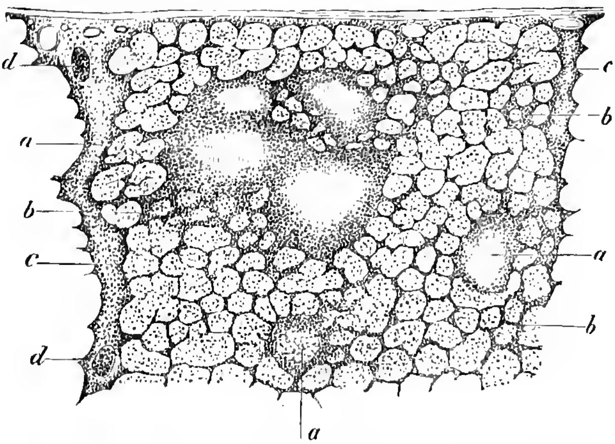


FIG. 445. CASEOUS LOBULAR TUBERCULOUS BRONCHOPNEUMONIA.

(*Section through a subpleural lobule; preparation hardened in alcohol, and stained with haematoxylin:* × 25)

a nodule with caseous centre and cellular periphery
b alveoli filled with exudation, the walls thickened and infiltrated with leucocytes
c interlobular septa infiltrated with leucocytes
d lymphatics filled with exudation

In the early stages of the process the grey infiltrated lobules have often a gelatinous appearance on section, and this is by some referred to as gelatinous infiltration. The alveoli are filled with liquid and cells (Fig. 445 *b* and Fig. 446 *c*), and occasionally with fibrin, while here and there the interlobular septa are infiltrated with leucocytes (Fig. 445 *c* and Fig. 446 *a*).

The structure of the pulmonary tissue within the primary foci breaks down in consequence of the inflammatory proliferation (Fig. 445 *a*); but in the parts that are merely infiltrated with the inflammatory exudation (*b*) the details of its texture can usually be recognised even after caseation has taken place.

Caseous lobular bronchopneumonia is sometimes the predominant or the only variety of the affection that is apparent in a tuberculous lung; but more commonly it involves only a limited portion of the organ, being accompanied in other parts by the fibro-caseous nodular variety. It may indeed be present at one part or another in every form of chronic pulmonary tuberculosis; for it is the rule rather than the exception to find several varieties of tuberculous lesion in one and the same lung. Caseous nodose and lobular bronchopneumonia is least often accompanied by disseminated nodular indurations in the case of young children, and was formerly wont to be described as **scrofulous pneumonia**.

The larger bronchi as well as the smaller bronchioles are often gravely affected in the caseous, in the indurative, and in the mixed forms of tuberculosis. The bronchial walls and the peribronchial connective tissue are at times thickly beset with tubercles, and the whole is at length converted into a continuous caseous mass.

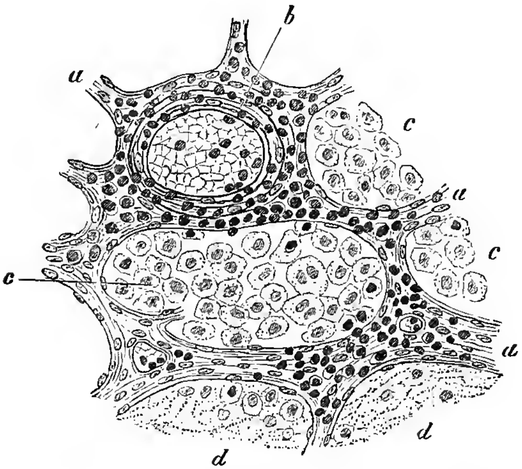


FIG. 446. LOBULAR BRONCHOPNEUMONIA IN PROCESS OF CASEATION.

(*Preparation hardened in alcohol, and stained with haematoxylin:* × 120)

a interalveolar septa infiltrated with cells
b vein with infiltrated wall
c alveoli filled with large epithelioid cells
d alveolar contents consisting of granular and filamentous fibrin

The pleura is always involved when secondary subpleural lesions are formed, and most notably in the nodose and the lobular caseous varieties, the serous membrane becoming covered with a fibrinous exudation. In the course of time the fibrinous deposit is replaced by new fibrous tissue, which gives rise to thickening of the pleura or adhesions between its apposed surfaces.

Caseation and disintegration of a subpleural tuberculous patch often extend to the pleura overlying it, and sometimes lead to its perforation. When air is thus permitted to enter the pleural cavity from the lung, **pneumothorax** is the result; and if the contents of a vomica thus escape into the cavity suppurative pleurisy is induced, giving rise to what is known as **pyopneumothorax**. By the liquid effusion the lung is compressed, and if its adhesions permit it may be forcibly displaced towards its hilum and the vertebral column.

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269. Inflammations of the lung due to **syphilis** are rare, and when they occur they do not always give rise to anatomical changes that can definitely be recognised as syphilitic.

It is well known that the syphilitic virus, when it gains access to the blood, is capable of inducing in the tissues forms of inflammation that either differ little from non-specific forms, or are distinguished by their tendency to assume a gummatous character. Both forms of inflammation are met with in the lung, but (if we exclude congenital cases) they are very rare indeed, and it is generally difficult to demonstrate their syphilitic nature and origin by histological methods.

Gummatous nodes are circumscribed lesions of the lung taking the form of patches of caseous granulation-tissue surrounded by a zone of inflamed alveolar parenchyma or of firm hyperplastic fibrous tissue. Such patches have often been described, but it is certain that in many cases they are not really of syphilitic origin.

Gummatous nodes of the lung are more frequently met with in infants affected with congenital syphilis than in adults; in the former they are sometimes very numerous. When recent they appear as greyish-red or greyish-white semi-translucent nodes varying in size from that of a pea to that of a hazel-nut. Later on they become opaque and white in the centre, and as they gradually break down they sometimes become excavated into small vomicae.

Another lesion attributable to congenital syphilis is the **syphilitic pneumonia** of infants, in which the pulmonary tissue over a varying extent undergoes proliferous multiplication of its fibrous constituents (Fig. 447 *a*), and multiplication and desquamation of its alveolar epithelial cells. The affected part may consist simply of alveolar tissue whose septa are somewhat thickened (*e* e_1), and when the infant has breathed the alveoli still contain air. In other parts that are dense and firm the alveolar structure is imperfectly developed, the foetal condition being persistent, and the compact tissue (*a b*) contains merely a few ducts and acini (*d* d_1) more or less imperfectly lined with epithelium, and is infiltrated with proliferous cells and leucocytes. The tissue is usually anaemic and its arteries have thickened walls, the adventitia in particular being hyperplastic (*c*).

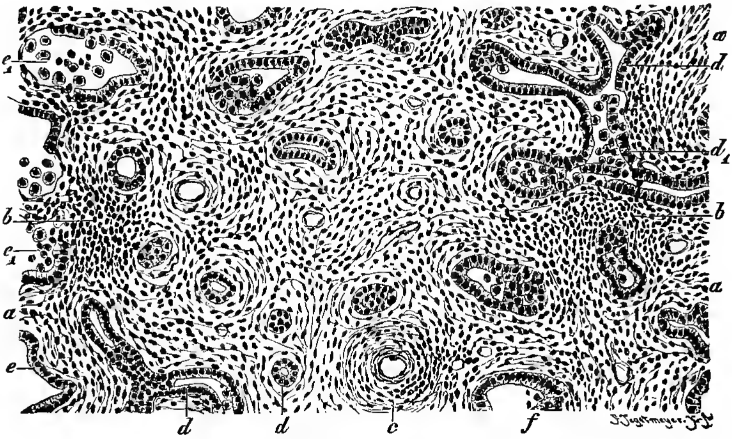

FIG. 447. CONGENITAL PULMONARY SYPHILIS.

(*Preparation hardened in Müller's fluid, stained with haematoxylin and eosin, and mounted in Canada balsam*: × 40)

a proliferous compact tissue of the undeveloped lung
b patches of granulation-tissue
c artery with thickened adventitia
d rudimentary bronchial ducts containing leucocytes and desquamated epithelium (d_1)
e rudimentary alveolar acini containing leucocytes and desquamated epithelium (e_1)

The histological appearances indicate that the affection is essentially due to morbid proliferation of the pulmonary fibrous tissue, associated it may be with inflammatory changes, and with epithelial proliferation and desquamation in the bronchioles and alveoli or their foetal representatives.

The disease has been called **white pneumonia**, on account of the dull-white or greyish appearance of the affected tissue. This appearance is seen both in the air-containing tissue and in the airless portions. Some authors however restrict the term to a morbid condition of the lung, also met with in syphilitic infants, in which the white tint and the consolidation are due in large measure to the accumulation within the alveoli of fatty desquamated epithelial cells. The diffuse and the nodose varieties of syphilitic inflammation and hyperplasia are sometimes co-existent in the same lung.

According to certain authorities, diffuse syphilitic inflammation of the lung may also supervene in adults, as the result of acquired syphilis, and occasionally leads to fibroid induration of its parenchyma. According to Pankritius, the inflammation starts from the hilum of the lung and extends radially into the pulmonary tissue. Others have described as syphilitic cases in which the indurative inflammation appears to have started from the pleura or the interlobular septa.

Some of the fibroid indurations discovered in the lungs of patients that are known to be syphilitic may possibly have been induced by the specific virus, but it must be extremely difficult even in these cases to determine the point with certainty. It is at any rate beyond doubt that many cases of pulmonary induration or cirrhosis described as syphilitic are in no way due to the disease, but are brought about by other causes; and the like is true of many so-called syphilitic cicatrices and thickenings of the pleura or of the interlobular fibrous tissue.

According to some, syphilitic catarrhal bronchopneumonia is an occasional result of syphilitic bronchitis. The bronchopneumonic patches either recover or issue in fibroid induration of the lung.

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Campana: *Dei morbi sifilitici e venerei* Genoa **1889**
Colomiatti: *A. f. Derm. u. Syph.* v **1878**
Councilman: Syphilis of the lung *Johns Hopkins Hosp. Bulletin* II Baltimore **1891**
Gerhardt: *Sitzungsber. d. phys.-med. Gesellsch.* Würzburg **1881**
Hecker: *V. A.* 17 **1859**; *Verhandl. Berlin. geburtschülfl. Gesellsch.* VIII **1854**
Heller: Affections of the lung in congenital syphilis *D. A. f. klin. Med.* XLII **1887**
Hiller: Pulmonary syphilis and phthisis *Cent. f. med. Wiss.* **1884**; *Charité-Ann.* IX **1884**

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LANG: *Vorles. über Path. und Therap. d. Syphilis* Wiesbaden **1895**
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PANKRITIUS: *Ueber Lungensyphilis* Berlin **1881**
PAVLIKOFF: Pulmonary syphilis *V. A.* 75 **1879**
PORTER: Relation of syphilis to phthisis *New York Med. Journ.* XLII **1885**
RAMDOHR: Pulmonary syphilis in adults *A. d. Heilk.* XIX **1878**
SCHNITZLER: *Die Lungensyphilis* Vienna **1880**
SCHÜTZ: Syphiloma of the lung *Klebs' Beiträge z. path. Anat.* part 1 **1878**
SPANUDIS: Congenital syphilis of the lung *Inaug. Diss.* Freiburg **1891**
STRÖBE: Histology of congenital syphilis of the lung *Cent. f. allg. Path.* II **1891**
VIERLING: *D. A. f. klin. Med.* XXI **1877**
VIRCHOW: *V. A.* I **1847** and 15 **1858**, and *Die krankhaften Geschwülste* II **1865**

270. **Actinomycosis** of the lung appears to be always due to aspiration of the specific fungus (*Actinomyces*) into the bronchi and their ramifications, if we exclude cases in which the infection has spread by direct extension downwards from the mouth and pharynx to the pleura and so to the lung.

According to J. ISRAEL, the symptoms are those of superficial catarrh of the mucous membrane of the air-passages, with a foetid tenacious secretion containing the characteristic fungus-clusters or grains. When the fungus reaches the pulmonary parenchyma it excites in it destructive and plastic inflammatory changes. Around the point of settlement nodules of vascular granulation-tissue are produced (Fig. 448 *b*), within which the characteristic fungus-clusters (*a*) are developed. When recent the granulomatous nodules are grey or greyish-red; but in general many of the cells become fatty and impart a yellowish-white tint to the nodule, while immediately around the fungus-clusters yellowish-white pus is apt to collect.

As the fungus spreads through the lung the nodules of granulation-tissue become more numerous, and in this manner red, greyish-red, or grey pneumonic patches are developed, each enclosing a certain number of small yellowish-white foci of from one to three or four millimetres in diameter, which yield on pressure a small amount of pus and little spherical clusters of *Actinomyces* (Fig. 448 *a*). By the radial extension and coalescence of these foci of disintegration large cavities are occasionally produced, each containing a yellowish pulp composed of pus-corpuscles, fat-granule cells, oil-globules, broken-down blood-corpuscles, and rounded tufts of *Actinomyces*. In other cases no such excavations are formed, and the process takes on an indurative character, each granulomatous nodule (*b*) becoming encapsuled in coarse fibrous tissue (*c*).

The new fibrous tissue developes not only in the septa but also within the alveoli (*g h*), where it is elaborated by fibroblasts that collect in the alveolar cavities and by capillary offshoots (*h*) from the adjacent blood-vessels.

The fibrous hyperplasia is always accompanied by exudative inflammation, leading to cellular infiltration around the blood-vessels (*i*), to distension of the alveoli with liquid containing leucocytes and proliferous desquamated epithelium (*d*), and in many places to fibrinous or croupous consolidation.

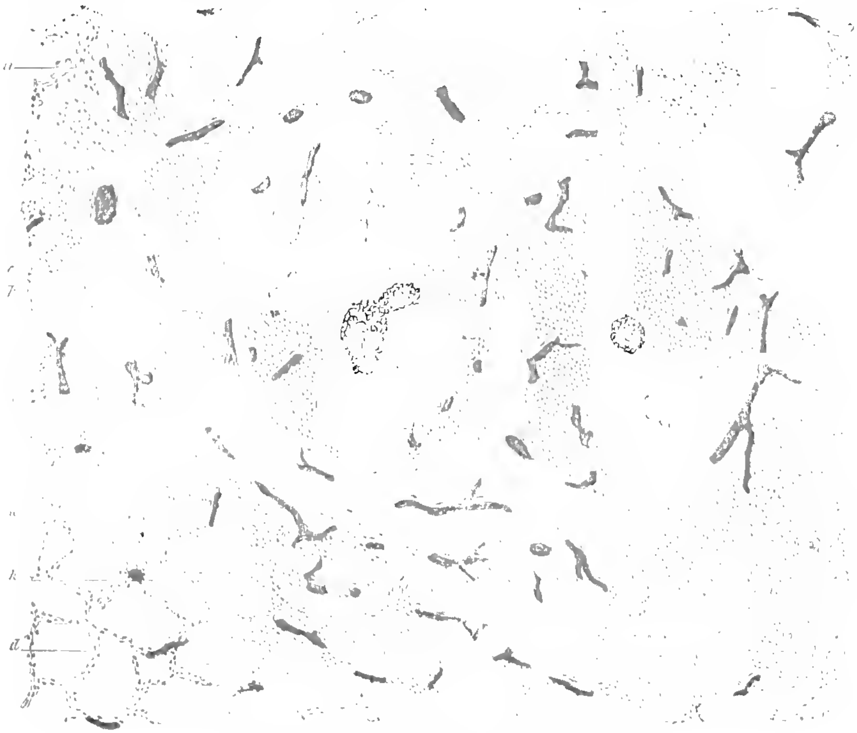

FIG. 448. ACTINOMYCOSIS OF THE LUNG.

(*The actinomyces-filaments have been stained with gentian-violet and are amplified more than the remaining portions of the drawing: preparation hardened in Müller's fluid, stained with carmine, and mounted in Canada balsam:* × 45)

a	fungus-cluster
b	granulomatous nodule composed of small round-cells
c	fibrous tissue
d	alveoli filled with large and small cells
e	bronchiole whose wall is infiltrated with cells
f	granulomatous patch in the neighbourhood of the bronchiole
g	alveoli filled with vascular fibrous tissue
h	fibrous tissue growing into the alveoli
i	normal arterioles
k	arterioles of the inflamed region

The bronchi within the affected region are also inflamed, their walls (*e*) and the connective tissue (*f*) surrounding them being often densely infiltrated with cells.

As the separate patches thus expand, and by the transport of the fungi new patches are produced that coalesce with the old ones, the greater part or the whole of a lobe may in a few weeks or months be converted into coarse shrunken cicatricial tissue, white, grey, or mottled with black, and enclosing a number of small yellow nodules with softened centres, or a few large cavities filled with yellow pus-like detritus. If the disease is still in active progress the tissue about the primary patches is studded with small indurated secondary nodules.

The disease may start in any part of the lung, and extend from this as a centre, the process being gradually arrested at the point of origin as the lung-tissue becomes fibroid and shrunken, while it still advances at the periphery. Sooner or later the pleura becomes involved, and pleuritic effusion or fibrous hyperplasia takes place according to the intensity of the resulting inflammation. The hyperplasia causes thickening of the serous membrane and firm adhesions between the apposed surfaces overlying the pulmonary lesions, and sometimes for some distance beyond them.

When the matted clusters of *Actinomyces* reach the bronchi from one of the cavities, they make their appearance in the sputum. Should some of the fungi be aspirated into previously healthy portions of the diseased lung or into the other lung, the result is a secondary infection, causing a fresh eruption of nodules of different sizes. These pass through the same changes as the primary foci, and either break down by softening or become converted into slate-coloured fibroid patches enclosing numerous little foci of granulomatous infiltration and disintegration.

From the pleura the disease at times extends to the muscles of the thoracic wall, the subcutaneous tissue, the skin, pericardium, mediastinum, diaphragm and underlying retro-peritoneal tissue, and so into the abdominal cavity. Wherever the ray-fungus thus settles it induces granulomatous proliferation, and the resulting morbid tissue sooner or later becomes fatty, undergoes the characteristic form of suppuration, and breaks down into large irregular sinuous abscesses. The tissue surrounding these abscesses is converted by fibrous hyperplasia into a coarse and thickened cicatricial envelope. When the skin is broken through, fistulous pus-secreting tracks remain, and communicate with cavities filled and lined with ragged yellowish friable granulations and detritus.

Similar changes take place in the mediastinal and retro-peritoneal tissues: in the pericardial sac necrotic granulations and dirty puriform exudations are formed.

Glanders of the lung gives rise to the formation of small greyish or yellowish cellular nodules varying from the size of a millet-seed to that of a pea, or to diffuse greyish or purulent infiltration, abscesses, lobular and lobar pneumonic consolidation, and circumscribed patches of haemorrhagic infiltration. Up to the present the channel of infection has not been definitely made out; it is probable however that the specific bacilli reach the pulmonary parenchyma both by way of the blood and with the inspired air.

In **leprosy** the lungs are very frequently affected. The morbid appearances closely resemble those of some varieties of chronic tuberculous disease, and in particular the fibro-caseous bronchopneumonic form (Art. 267).

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CROOKSHANK: Actinomycosis *Lancet* I **1897**
DELÉPINE: Leprosy *Trans. Path. Soc.* XLII London **1892**
DIECKERHOFF: *Lehrb. d. spec. Path. f. Thierärzte* I Berlin **1885**
ISRAEL, J.: *Klin. Beiträge zur Kenntniss der Aktinomykose des Menschen* Berlin **1885** (trans. New Syd. Soc.) London **1886**; *Cent. f. med. Wiss.* XXIV **1886**; *A. f. klin. Chir.* XXXIV **1886**
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LINDT: Primary actinomycosis of the apex of the lungs *Correspbl. f. Schweizer Aerzte* **1889**
MOOSBRUGGER: Human actinomycosis *Beiträge von Bruns* II **1886**
PFLUG: Actinomycosis *Oesterreich. V. f. wiss. Veterinärkunde* 58 **1882**; Glanders *Zur pathol. Zootomie des Lungenrotzes der Pferde* Leipzig **1877**
PUSCH: *A. f. Thierheilk.* IX **1883**
PÜTZ: *Seuchen und Herdekrankheiten* Stuttgart **1882**
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CHAPTER LXXXVII

TUMOURS AND PARASITES OF THE LUNG

271. **Carcinoma** is the most common of the primary tumours of the lungs and bronchi; it may develope from the lining epithelium of the bronchi or of the alveoli. It usually takes the form of large soft solitary nodes of a white or reddish-white colour, which infiltrate the adjacent tissues and give rise to metastases in the lymphatics and bronchial glands.

In the larger bronchi carcinoma produces papillary or tuberous excrescences, which break through into the contiguous lung-tissue. Carcinoma arising from the alveoli or from the bronchi, and straightway invading the peribronchial lymphatics, is apt to spread rapidly within these channels, with the result that the lung is soon studded with small peribronchial and interlobular marrow-like nodules.

Chiari has described an **adenoma** of the mucous glands in the bronchial mucous membrane, but this neoplasm is very rare.

Rokitansky, Morgan, Rindfleisch, and others have described cases of **fibroma**, in which nodules from the size of a hemp-seed to that of a hazel-nut were formed in large numbers around the bronchi. **Osteoma** also occurs in the form of irregularly-shaped fragments with jagged processes, and of rounded nodules of the size of a pea; small globular **lipomata** (Rokitansky, Chiari), **chondrolipomata**, and rounded **enchondromata** starting from the bronchial cartilages, have also been met with. The jagged or spinous osteomata have in a few rare instances been found in large numbers within the lung.

Of **secondary growths** examples of each species of neoplasm that forms metastases at all have been found in the lung. When the tumour-cells reach the lung as emboli they usually produce rounded nodules having the characters of the parent-tumour. These start from the embolised blood-vessels, and grow by radial extension or concentric accretion, partly invading and partly compressing the pulmonary tissue.

When the tumour-cells originally reach the lung or pleura by way of the lymphatics, nodules of various sizes appear along the course of the latter. In the case of carcinoma the metastases are often diffused with remarkable uniformity (Fig. 449 *c*), the lymphatics of a large portion or the whole of the lung being all distended with white marrow-like masses. On section such a lung exhibits a number of close-set whitish or reddish nodes and ridges along the course of the bronchi (*b*), arteries (*a*), or interlobular septa.

As the tumour grows reactive inflammation is apt to be set up, especially in the pleura, where it not infrequently assumes a haemorrhagic character: in the lung the inflammation is usually catarrhal (*d*).

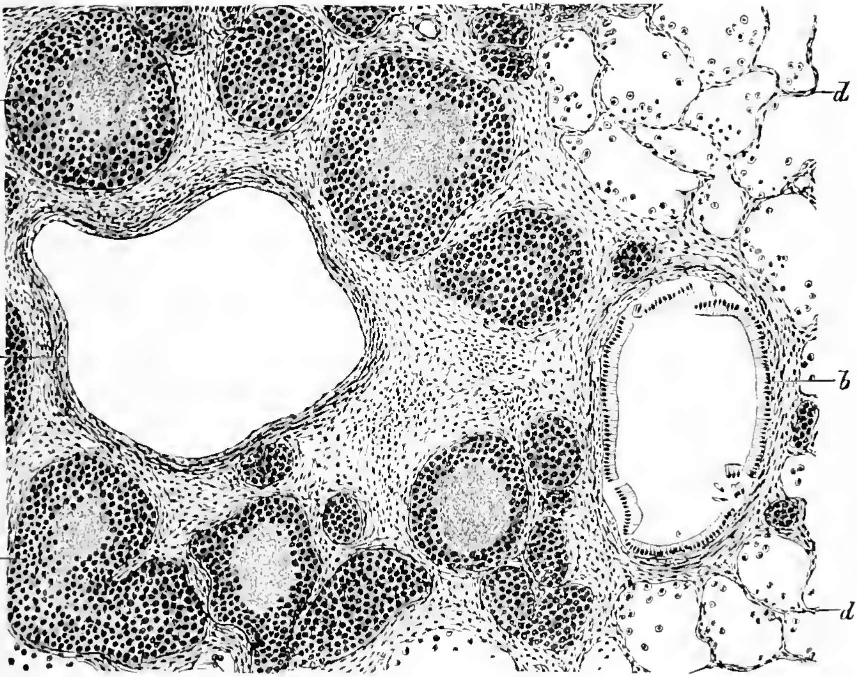

FIG. 449. METASTATIC CARCINOMA OF THE LUNG.

(*Section of a growth secondary to carcinoma of the stomach, involving the periarterial and peribronchial lymphatics: hardened in Müller's fluid, stained with haematoxylin and eosin, and mounted in Canada balsam:* × 25)

a artery
b bronchus
c periarterial and peribronchial lymphatics filled with cancer-cells and lymph
d alveoli containing desquamated epithelium and leucocytes

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CHIARI: Various tumours *Prager med. Woch.* **1883**
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Finlay and Parker: Primary cancer *Med.-chir. Trans.* LX London **1877**
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van Gieson: Carcimona *New York Med. Record* XVI **1879**
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Hertz: Tumours *Ziemssen's Cyclop.* v New York **1875**
Hesse: Primary cancer *A. d. Heilk.* XIX **1878**
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Ribbert: Lymphoma *V. A.* 102 **1885**
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Rütimeyer: Primary sarcoma of the lungs *Correspbl. f. Schweizer Aerzte* **1886**
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Shattuck: Tumours *Ziemssen's Cyclop.* (supplement) New York **1881**
Siegert: Tumours of the lower air-passages (papilloma and chondroma) *V. A.* 129 **1892**; Histogenesis of primary cancer of the lung *ibidem* 134 **1893**
Stilling: Primary bronchogenous carcinoma *V. A.* 83 **1881**
Virchow: *Die krankhaften Geschwülste* II
Weichselbaum: Adenosarcoma *V. A.* 85 **1881**
Werner: Primary carcinoma of the lung *Inaug. Diss.* Freiburg **1891**
West: idem *Trans. Path. Soc.* XXXV London **1883**

272. The **animal parasites** infesting the human bronchi and lungs are not numerous. The most important is *Echinococcus*, which may form hydatid cysts of considerable size, with or without daughter-cysts. *Cysticercus cellulosae* is rare. *Strongylus longevaginatus*, a cylindrical worm 15–26 millimetres long, has once been found in a boy's lung. In several cases of gangrene of the lung Kannenberg discovered among the shreds of lung-tissue in the sputum *Monas lens* and *Cercomonas*, two flagellate infusorians. In the resting-state they look not unlike colourless blood-corpuscles.

Of the **vegetable parasites** of the lung the most noteworthy are the numerous varieties of bacteria. Some of these, such as the bacilli of tuberculosis, anthrax, and glanders, actinomyces, and the pneumococcus, give rise to specific inflammations. Other bacteria, such as those that inhabit the mouth, may cause acute non-specific inflammations of various degrees of intensity when aspirated into the air-passages.

In tuberculous cavities, disintegrating haemorrhagic patches, croupous exudations within the bronchi, trachea, etc., we occasionally meet with a small *Sarcina*. It usually occurs at the same time also in the pharynx and larynx, but has probably no causal relation to any of the diseases in question.

Of the filamentous fungi or *Hyphomycetes* we find in the lung various forms of *Aspergillus* and *Mucor*, and *Oïdium albicans*, which settle only in decomposing lung-tissue, stagnant secretions, or haemorrhagic infiltrations. The above-named moulds now and then proceed to the stage of fructification within the lung. Affections of the lung due to the settlement and multiplication of colonies of fungi are known as **pneumonomycoses.**

According to BAELZ (*Cent. f. med. Wiss.* no. 39 **1880**), a peculiar parasitic disease of the lung (*gregarinosis pulmonum*) is very common in Japan. Patients affected with it spit blood for a number of years, and their lungs contain encysted brownish-yellow ovoid *Psorospermia*, and non-encysted clear or pale-yellow granular round or ovoid *Coccidia*. A like affection is also met with in Formosa, and according to MANSON is due to the presence in the lung of *Distoma ringeri*, of which BAELZ'S *Psorospermia* are said to be merely the ova (*Med. Times and Gazette* II **1881**, II **1882**; *B. M. J.* II **1882**).

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VON BESSER: The bacteria of the normal air-passages *Ziegler's Beiträge* VI **1889**
BOECKL: *D. Z. f. Thiermed.* X **1884**
BOLLINGER: *Zur Aetiologie der Infectionskrankheiten* Munich **1881**
FALKENHEIM: Sarcinae *A. f. exp. Path.* XIX **1885**
FISCHER: Sarcinae *D. A. f. klin. Med.* XXXVI **1885**
FRIEDREICH: Sarcinae *V. A.* 30 **1864**
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GAFFKY: Sarcinae in phthisis *A. f. klin. Chir.* XXVIII **1882**
HAUSER: Sarcinae of the lungs *Fortschr. d. Med.* VI **1888**
NAUWERCK: Pneumonomycosis due to sarcinae *Correspbl. f. Schweizer Aerzte* XI **1881**
PODACK: Aspergillar mycoses *V. A.* 139 **1894** (with references)
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CHAPTER LXXXVIII

THE PLEURA

273. The **pleura** is a thin fibrous membrane covered with a single layer of large flattened cells, which lines the thoracic cavity and invests the lungs. The morbid changes to which it is subject are usually of a secondary nature, being consecutive to morbid conditions of the lung, the bronchial glands, the mediastinal tissue, and the bony and other structures enclosing the thoracic cavity.

In affections accompanied by great venous engorgement of the thoracic contents, in haemophilia, and in certain infective diseases, small **haemorrhages** into the pleura and subpleural tissue sometimes take place. Numerous petechiae are generally found in the pleura and subpleural tissue in cases where death has resulted from suffocation. These extravasations are due to the excessive accumulation within the thoracic vessels of blood that is as it were pumped into them, when violent inspiratory efforts are made and air is at the same time prevented from entering the chest freely.

Haemorrhage into the pleural cavity is usually the result of traumatic injury of the lung, rupture of an aortic aneurysm, or tuberculous and cancerous disease. When the structure of the pleura is not already altered by disease, the blood is re-absorbed as in the case of haemorrhagic effusion into the peritoneal cavity. Adhesion of the layers of the pleura is a common result of the absorption of large quantities of coagulated blood. **Haemopneumothorax** is the condition in which, when the lung or the chest-wall is wounded, air enters the pleural cavity with the effused blood. When the air is aseptic it is absorbed from the cavity without inducing inflammation.

Oedema from cardiac or renal disease, due to vascular engorgement and textural changes in the vessel-walls, frequently gives rise to serous effusion into the pleura. The liquid is clear and yellowish in colour, and as it accumulates in the pleural sac it compresses the lung against the spine: the condition is known as **hydrothorax**. When pleural adhesions already exist, the liquid saturates the adhesive membranes and gathers in the intervening spaces (circumscribed, sacculated, or multilocular hydrothorax). Laceration or rupture of the thoracic duct sometimes allows chyle to escape into the thoracic cavity, the result being described as **chylous hydrothorax**.

Inflammation of the pleura, or **pleurisy**, is usually associated with inflammation of the lung, thoracic wall, pericardium, or peritoneum, with traumatic injuries, or with certain infective and toxaemic conditions such as acute articular rheumatism, infective nephritis, pyaemia, typhoid fever, gout, and the acute exanthemata. At times indeed pleurisy is the only local indication that infection has taken place. The inflammation of the pleura that invariably accompanies croupous pneumonia is probably in some cases due to the same cause as the pulmonary inflammation and is thus co-ordinate with it; in other cases the pleurisy is no doubt consecutive and due to secondary infection from the lung. The resulting effusion may thus contain both pneumococci and streptococci. In some cases of pleuro-pneumonia the pleurisy is the more prominent affection of the two.

Metastatic croupous, haemorrhagic, purulent, or gangrenous circumscribed pneumonia, and all the various forms of bronchopneumonia, when the pulmonary lesions they induce are subpleural, invariably affect the pleura to some extent. Pulmonary tuberculosis likewise sooner or later sets up pleural inflammation, the infection of the membrane being due either to the tuberculous virus itself or to pyogenic micrococci. Inflammations of the pericardium or peritoneum may extend to the adjacent portions of the pleura. Ulcers of the stomach and oesophagus, abscesses of the liver and spleen, perityphlitic retro-peritoneal abscesses, tuberculous foci in the periosteum of the vertebrae or of the ribs, and mediastinal inflammations, are all likewise capable of giving rise to pleurisy. The actual exciting causes of the inflammation are generally pneumococci, staphylococci, or streptococci.

The inflammatory exudations are in many cases of a purely fibrinous character (dry pleurisy), and are deposited on the surface of the pleura in the form of small curds and flakes, or of yellowish-white films and membranes. Serous and sero-fibrinous effusions are somewhat less common, and contain filaments and flakes of fibrin, the pleura itself being covered over with a fibrinous deposit. Both kinds of exudation are met with in the primary as well as in the secondary forms of pleurisy.

Purulent effusion (purulent pleurisy, pyothorax, **empyema**) is commonly a concomitant of metastatic septic pneumonia, or follows the rupture of a tuberculous cavity in the lung or one of the bones, the irruption of a hepatic abscess, or the perforation of a carcinomatous ulcer of the oesophagus or stomach. The pyogenic infection may however reach the pleura without affecting the lung, either by way of the circulation or from a septic wound of the thorax: the microbes that convey the infection are the same as those that set up suppurative inflammation in other parts.

The exudation is either purulent from the outset, or beginning as a sero-fibrinous effusion it becomes turbid and puriform after some time. Pleuritic effusions set up by gangrene of the lung, or by perforation of a gastric or intestinal ulcer, are in general foetid and ichorous. A haemorrhagic exudation usually indicates the existence of tuberculous or cancerous inflammation, if we exclude cases of haemorrhagic purpura and scurvy.

Scanty fibrinous exudations are usually limited to circumscribed portions of the visceral pleura, and give a turbid appearance to the membrane. When more abundant they generally spread over the greater part of the pleura, and by extending along its lymphatics to the interlobular connective tissue may give rise to interlobular pneumonia (Fig. 435).

The amount of a pleuritic effusion varies from a few grammes to as much as three kilogrammes. The liquid accumulates, unless adhesions confine it in some way, in the lower part of the pleural cavity. As the liquid increases in amount the lung is subjected to greater and greater pressure, till at length it collapses into a compact shrunken airless mass, grey, greyish-black, or brownish in colour, and tightly pressed against the spine. At the same time the diaphragm is forced downward, the heart and the mediastinum are displaced to the opposite side, and the large thoracic vessels are compressed. The same conditions are produced when the pleural cavity is distended with air (pneumothorax), or with a mixture of pus and air (pyopneumothorax). Should pleural adhesions already exist when the effusion is poured out, the liquid accumulates wherever it can find room.

The **re-absorption** of serous exudations is in some cases rapid, in others somewhat slow. When much fibrin is deposited upon the surface of the pleura, it usually becomes organised and replaced by new fibrous tissue in the manner we have already described when treating of the pericardium (Art. 15). The result is thickening of the pleura and adhesion of its apposed surfaces (adhesive or plastic pleurisy).

Minute foci of proliferation of this kind lead to the formation of little white spots or flattened nodules on the surface; larger foci produce diffuse white thickenings and fibrous adhesions in the form of bands and membranes traversing the cavity and binding down the lung. Recurrent attacks of pleurisy, such as are not uncommon in pulmonary tuberculosis, often give rise to enormous thickening of the pleura, the parietal and visceral layers being inseparably fused into a single dense cicatricial mass, sometimes almost cartilaginous in consistence and from 0.5 to 2 centimetres thick, which is so firmly adherent on its costal aspect that it can scarcely be stripped from the thorax-wall.

When the re-absorption of the effusion is slow, or when fresh liquid is continually being poured out, and close adhesion of the apposed surfaces is thereby prevented, a large portion of the visceral layer is often converted into leathery cicatricial tissue (Fig. 436), which sometimes sends processes into the interlobular fibrous tissue of the lung. As this shrinks the lung becomes permanently contracted and distorted, and its free edges rounded and blunt. The space left vacant by the contraction of the lung is at first occupied by liquid, but as this is absorbed the volume of the thoracic cavity on the affected side is gradually diminished by displacement of the mediastinum and its contents, by elevation of the diaphragm, by contraction of the chest-wall and imbrication of the ribs, and by lateral curvature of the spine. The cicatricial adhesions occasionally become calcified.

When the exudation is purulent or putrid, portions of the pleura are apt to soften and break down by erosion. Should the visceral layer thus give way, pus gains entrance into and infiltrates the lung, and passing thence into the bronchi is evacuated by coughing and expectoration. The place of rupture in the pleura and the purulent infiltration of the underlying lung-tissue are often discoverable *post mortem;* though there is usually no direct opening by which air can gain access from the bronchi to the pleural cavity. But if an abscess of the lung has burst into the pleura, it is often possible to trace with the probe a communication between the pleural rent and a bronchus. Pyopneumothorax is therefore common in the latter case and rare in the former.

If the suppurative softening extends to the parietal layer of the pleura, pus gradually forces its way between the ribs till it reaches the subcutaneous tissue and gives rise to an intercostal abscess: such abscesses generally point between the costal cartilages near the sternal articulations.

In a few rare instances pleuritic suppuration extends to the mediastinum, the pericardium, the peritoneum, or the retro-peritoneal tissue. From the mediastinum the suppuration may spread to the pleura of the opposite side.

Recovery from empyema takes place when the pus contained in the pleural cavity is evacuated spontaneously or by surgical operation, the pleura then becoming lined with a layer of granulation-tissue which long continues to secrete pus. By and by the granulating membrane becomes converted into a dense cicatricial envelope, while the cavity of the empyema is sooner or later filled up partly by new fibrous tissue, partly by the falling in of the thorax-wall and the displacement towards it of the diaphragm, the lung, and the mediastinum with the pericardium. The younger the patient the more readily does the thorax fall in and the spine curve over to the affected side.

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274. **Tuberculosis** of the pleura is a result either of haematogenous miliary tuberculosis or of direct extension from tuberculous lesions of the lung, the bronchial lymph-glands, the bones of the thorax, the diaphragm, or in rare cases of other neighbouring organs. Tubercle-bacilli sometimes reach the pleura without infecting any other structure by the way, and so induce primary tuberculous pleurisy: the tubercles thus produced in the membrane may or may not be surrounded by a hyperaemic areola.

The inflammatory process set up by tubercle-bacilli that have gained direct access to the pleural sac is usually somewhat intense, and is accompanied by haemorrhagic sero-fibrinous effusion and presently by the production of granulations containing tubercles. Should necrotic detritus from a tuberculous lesion of the lung or a tuberculous vertebral abscess enter the pleural cavity, or should air be admitted into it at the same time, the resulting effusion is usually purulent, and empyema or pyopneumothorax is the result.

Pleurisy consecutive to pulmonary tuberculosis generally results in the formation of fibrous adhesions that are free from tubercles; but sometimes the inflammatory hyperplasia is more abundant, and highly-vascular fibrous membranes and bands are elaborated containing multitudes of discrete and agglomerate tubercles, and accompanied by sero-haemorrhagic, fibrino-haemorrhagic, or in some cases simply haemorrhagic effusions. In chronic cases the agglomerate tubercles are apt to be converted into caseous nodes enclosed in coarse fibrous tissue.

The infection of the pleura from tuberculous lesions of the spine or of the bronchial lymph-glands is manifested either by a more or less copious eruption of tubercles in the neighbourhood of the primary lesion, with a scanty effusion, or by abundant sero-fibrinous or haemorrhagic exudations with subsequent fibrous hyperplasia.

Among the **primary tumours** of the pleura may be mentioned endothelioma and ordinary sarcoma, fibroma, angioma, lipoma, and osteoma; but these are all rare. **Endothelioma** takes the form of diffuse white fibroid thickenings of the pleura, with a certain number of nodose swellings scattered through them; it may appear on any of the parietal, pulmonary, or diaphragmatic surfaces. Its coarse fibrous stroma encloses nests and reticulated strings of cells that give it an appearance not unlike that of a hard cancer.

Secondary growths usually arise by metastasis from carcinoma of the breast, thyroid gland, oesophagus, or stomach, and take the form of strings of cancerous nodules of various sizes following the course of the lymphatics. If the nodular eruption is exceptionally abundant, it is not infrequently accompanied by sero-fibrinous and haemorrhagic exudations.

Of **animal parasites** *Echinococcus* is almost the only one that invades the pleura. The hydatid disease it occasions is sometimes primary in the pleural cavity; in other cases it is secondary to the irruption of hydatids from the lung or the liver.

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CHAPTER LXXXIX

THE THYROID GLAND

275. The adult **thyroid gland** consists of two lateral lobes and an isthmus uniting them across the front of the trachea. The vertical diameter of a lateral lobe is 5 to 7 centimetres; the breadth 3 to 4 centimetres; the width of the isthmus varies from 4 to 20 millimetres. Very frequently there is a middle lobe or pyramid which rises from the isthmus and grows upward. The parenchyma consists of a vascular fibrous stroma including in its meshes rounded follicles, which either possess a central lumen or are filled up entirely with small epithelial cells. In the former case the follicle is simply lined with cubical or cylindrical epithelium. In the later years of life some of the glandular follicles usually contain colloid matter.

Absence of the thyroid is rare. More common **anomalies** are—abnormal smallness or absence of a lobe or of the isthmus, abnormally large size (congenital goitre), multiple lobes, and accessory glandular masses separate from the main mass and connected with the hyoid, the deeper parts of the trachea, the supraclavicular fossae, the interior of the larynx, the aorta, or the posterior wall of the pharynx. In very rare instances the isthmus is found to pass between the trachea and the oesophagus.

In old age the substance of the gland undergoes more or less marked **atrophy,** the follicles shrinking to clusters of small cells or in places disappearing altogether, and the fibrous stroma becoming homogeneous and indurated, and at the same time increasing in relative amount. Premature atrophy has been observed in the case of cretins (HANAU) and of adults suffering from myxoedema.

The most important of the morbid changes to which the thyroid is liable are those forms of enlargement of the whole gland or of particular parts of it included under the general term **goitre,** bronchocele, or thyreocele (*struma*).

The gland may be of abnormal size from birth (congenital goitre), or it may become enlarged during childhood, at the time of puberty, or later on in life. The goitrous tumour is in some cases due to the uniform enlargement of one lobe or of the whole gland, in other cases to the development of large nodose masses within it.

Having regard to the clinical course and anatomical features of the affection, we may distinguish three forms of goitre: of these, two result from hyperplasia, and the third is due to arterial congestion or venous engorgement. The hyperaemic enlargement is transient, and might be described as **vascular goitre**.

Enlargements of the thyroid are sometimes produced by neoplasms like carcinoma and sarcoma, sometimes by growths approximating in appearance to the normal structure of the gland. The former class are generally regarded as cases of **malignant goitre**, the latter as innocent or benign.

Simple or **benign goitre** is due to hyperplastic proliferation of the glandular parenchyma, and different varieties of the affection are produced according to the distribution of the hyperplasia and the secondary changes that ensue in the overgrown tissue. Thus the overgrowth may be diffuse or nodose: in the former case the goitre is uniformly enlarged or smoothly lobate, in the latter it is tuberous and irregular, and both forms may reach the size of an infant's head. The enlargement may be unilateral or bilateral, or may even be limited to the isthmus.

The glandular proliferation results in the production of globular, ovoid, or cylindrical clusters of cells (Fig. 450 *a*), or of gland-like acini and tubules lined with epithelial cells (*b*) and partly filled with colloid matter (*c*): sometimes indeed both kinds of formations are present in about equal proportion.

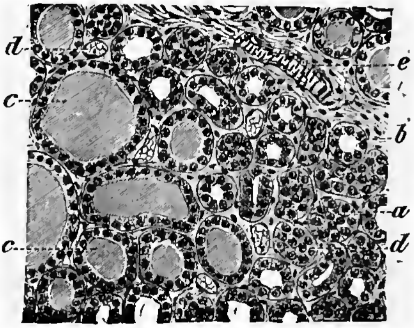

FIG. 450. GOITRE PARTLY HYPERPLASTIC AND PARTLY COLLOID.

(*Alum-haematoxylin staining*: × 8)

a	follicles filled with clusters of cells
b	follicle with its lumen unoccupied
c	colloid masses
d	capillaries
e	fibrous stroma with arteriole

When the goitre consists almost entirely of small solid clusters and columns of cells and of vesicles or acini devoid of colloid matter and separated by scanty more or less vascular fibrous tissue, its cut surface is of a whitish, dull-yellow, reddish-brown, or dark-brown colour, according to the amount of blood it contains. In parts where many of the acini have come to contain masses of colloid matter, the enlargement is correspondingly greater, and the section acquires a translucent honey-like appearance. When the production of colloid matter is excessive in amount and distends most of the acini, the tissue is reduced to a fine reticulum enclosing the translucent colloid matter in its meshes: in this case we have what is called colloid or **gelatinous goitre**.

In the parts where the intrafollicular colloid degeneration has reached its maximum, the goitrous tissue is entirely made up of large vesicles lined with a single layer of flattened cells, and separated only by the fibrous septa; but there are always some parts in which small empty acini and solid clusters of cells are interspersed among the large distended vesicles, and from these the hyperplastic process may start afresh.

Should the vesicles become over-distended by the continued accumulation of their colloid contents, the intervesicular septa usually become atrophic and break down, giving rise to the formation of large cyst-like cavities, which from their mode of origin we might term follicular cysts or cysts of distension. These cysts contain colloid matter, albuminous liquid, and desquamated fatty cells, their walls being lined with flattened or cubical epithelial cells, or in rare instances with columnar cells.

According to WÖLFLER glandular follicles lying close to a cyst of this kind occasionally push inwards and invaginate the cyst-wall, and with the aid of ingrowing connective tissue produce new vesicles in its interior. Papillary excrescences invested with columnar epithelium sometimes spring up from the inner surface of the cyst-wall (proliferous cystadenoma).

From the outset the vascular stroma also may become actively proliferous, and modify perceptibly the texture of the goitre. The blood-vessels, arteries, veins, and capillaries alike, undergo morbid multiplication, with the result that a **vascular goitre** is produced. If the capillaries are disproportionately dilated, the glandular follicles seem to be forced asunder by widely-distended blood-cavities, and the tissue gradually assumes the appearance of a telangiectatic or cavernous angioma with glandular follicles lying between the vessel-walls.

Goitres are extremely liable to internal **haemorrhage,** the blood saturating the parenchyma, and sometimes producing a notable enlargement of the tumour. After a very copious haemorrhage the infiltrated tissue is apt to become necrotic and break down into a brown or yellow greasy pulp. Should this be liquefied and absorbed, and the surrounding tissue become indurated by fibrous hyperplasia into a kind of capsule, a cyst containing coloured liquid takes its place. But if the disintegrated patch is not thus encysted, hyaline or fibrous and more or less vascular connective tissue grows into it by degrees, and in this tissue new cellular clusters, glandular follicles, and vesicular acini are occasionally reproduced.

The processes just described may give a fibrous appearance to the texture of the tumour, which has accordingly been described as **fibroid goitre.** The centre of the affected portion is usually converted into a dense indurated mass from which as a centre fibrous bands radiate towards the periphery. In some instances the fibrous hyperplasia extends more uniformly over the parenchyma, and causes fatty degeneration and atrophy of the glandular elements, while the new fibrous tissue itself tends to become denser and harder.

The interfollicular fibrous stroma not infrequently undergoes hyaline degeneration (Fig. 451), owing to the deposition of a hyaline substance between its fibrils, which subsequently swell up and become homogeneous (GUTKNECHT). The glandular cells (*b*) may remain intact for a while, but later on most of them break down, and their place is occupied by clear liquid (*c*). At the same time the walls of the capillaries and of the larger vessels also (*f*) undergo hyaline change, so that at length the tissue becomes non-cellular and homogeneous (*e*) or slightly turbid, with perhaps a few vacuoles scattered through it.

After the disappearance of the epithelial cells (*c*), all that is left of the affected part is a sponge-like structure of hyaline substance (*e*), with rounded meshes of various sizes containing a clear liquid. Blood is readily effused into these spaces when the goitre is wounded or bruised.

Where the hyaline transformation is complete the tissue of the goitre becomes translucent and almost mucoid in appearance (**myxomatous goitre**). Along the inner zone of the glandular cortex the jelly-like matrix is studded with white, yellow, or brown grains representing the degenerate follicles.

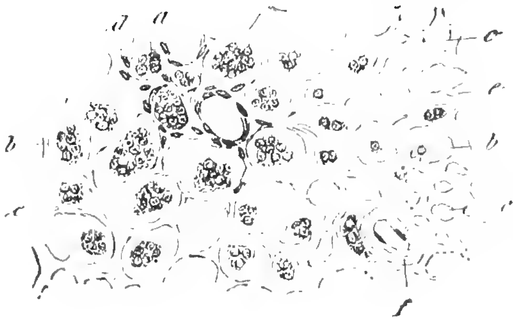

FIG. 451. HYPERPLASTIC GOITRE WITH HYALINE DEGENERATION OF THE STROMA AND ATROPHY OF THE EPITHELIUM.

(*Preparation hardened in Müller's fluid, stained with carmine, and mounted in Canada balsam*: × 30)

a follicle filled with epithelium
b follicle filled with epithelium and liquid
c follicle filled with liquid
d normal stroma with blood-vessel
e hyaline stroma
f blood-vessel with hyaline wall

The hyaline and indurated fibrous tissue often undergoes calcareous infiltration, and a fibroid or myxomatous goitre may thus become partially calcified and stony, forming what is known as **petrified goitre**. The colloid and other contents of the vesicles and cysts may in like manner become calcareous. In certain very rare cases the fibrous tissue undergoes **ossification** (FÖRSTER, LÜCKE).

Amyloid degeneration affects normal as well as goitrous glands, the vessels being the chief seat of the change. Some goitres also contain circumscribed amyloid deposits (BECKMANN); these take the form of lardaceous or wax-like nodes, and constitute what are called **waxy goitres**.

The hyaline and indurated stroma sometimes softens and breaks down in places, and cysts are thus produced as in the case of haemorrhagic infiltration. These cysts contain colloid matter, epithelial cells and detritus, blood, blood-pigment, cholesterin, and fat. The degeneration and disintegration are however limited to the central portions of the goitrous node, the walls of the resulting cyst being made up of the fibrous capsule of the node and of the still unsoftened glandular tissue. As the hyaline change in the stroma and the colloid degeneration and softening of the glandular tissue advance, the cysts are gradually enlarged. Intercurrent haemorrhages increase and modify their contents, and facilitate the disintegration of the surrounding tissue. In some cases the cyst-wall is thickened by the production in it of new fibrous tissue, but this often becomes hyaline and breaks down again: the cyst-wall is also liable to undergo partial calcification.

The numerous and diverse morbid changes to which the hyperplastic thyroid tissue is subject naturally have their effect on the appearance of the cut surface, which is remarkably heterogeneous and variable. Reddish-brown and dull-yellow patches of glandular tissue, and dark-red haemorrhagic spots, alternate with brownish or white-specked and fatty regions of softening, with colloid masses and tracts of translucent hyaline stroma devoid of gland-cells and saturated with liquid, and with lustrous white calcareous deposits. And when the goitre contains a number of nodes, not only do these vary in appearance among themselves, but the different parts of the same node are often in different stages and conditions of change.

A goitre that grows to any considerable size is apt to press upon and so to constrict the trachea, especially when both lobes are markedly enlarged and surround the tube, when the growth of one lobe forces it to the opposite side and indents it, or when a goitrous node grows down behind the manubrium. Very persistent and continuous pressure of this kind may at length cause atrophy of some of the tracheal cartilages.

Accessory thyroids, when they are present, are also liable to undergo goitrous enlargement. The **parathyroids,** two masses of epithelial tissue resembling the suprarenals in structure, and placed at the outer and inner borders of the lateral lobe, occasionally become hypertrophic.

The **aetiology** of non-malignant goitre is at present imperfectly understood. Frequently-repeated congestion of the gland seems to favour its development, but this alone is not sufficient to produce it. Probably the affection is due to something of the nature of an infection, though the infective agent is still unknown. In certain districts, even in particular villages or particular buildings (such as barracks and boarding-schools), goitre is endemic; and in these from time to time it spreads almost in epidemic fashion. The most natural inference would seem to be that the exciting cause is a miasma confined to particular localities.

It will probably be found that in most cases the exciting agent gains access to the body in drinking-water. This view is supported by the fact that families hitherto free from goitre acquire the disease when they migrate into districts where it is common, and conversely that goitrous patients may recover when they are removed to districts where it is unknown.

The general effects of goitre and other morbid conditions of the thyroid upon the rest of the body are dealt with in the volume on General Pathological Anatomy (Art. 22).

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276. Both epithelial and connective-tissue **neoplasms** are met with in the thyroid gland, and give rise to the form of enlargement known as **malignant goitre**. They differ from the benign enlargements of the gland in that they often extend beyond it and invade the neighbouring tissues, and that they produce metastases or secondary growths elsewhere.

The **epithelial** tumours, as a rule, take the form of soft marrowy nodose growths varying from the size of a hen's egg to that of a child's head, and situated in one of the lobes of the gland. They are usually surrounded by normal or hyperplastic gland-tissue. Rarely is the whole gland transformed into cancerous tissue. Secondary growths are generally produced. Irruption of the tumour into the trachea or larynx often takes place, and accordingly the tumour is much less movable over the underlying structures than an ordinary goitre. Malignant growths are especially apt to develope in glands that are already goitrous.

The structure of these tumours is most commonly that of typical carcinoma, but varieties are met with which may best be described as malignant **adenomata**. In rare instances adenomata occur that produce metastases, and these latter closely resemble the normal gland in structure. They however usually contain gland-like tubules and acini lined with tall columnar epithelium, within which papillary outgrowths sometimes spring up (*adenoma papilliferum*).

As a rule the cancerous growths have the structure of simple or of medullary carcinoma, the cancer-nests being made up of rounded or polymorphous cells. Squamous-celled carcinoma (epithelioma) is a very rare form (FÖRSTER, EPPINGER, LÜCKE, BRAUN, KAUFMANN).

Of the **histioid** or connective-tissue tumours of the thyroid **sarcoma** is the commonest, and like carcinoma usually arises in an already-existing goitre. Round-celled, spindle-celled, and polymorphous-celled varieties are described, and to the list of forms WÖLFLER adds giant-celled sarcoma, angio-sarcoma, and alveolar sarcoma. They form irregular nodulated tumours extending over a part or the whole of a single lobe, seldom over the entire gland.

The cut surface is generally smooth, though the tumour is often more or less lobulated by bands of fibrous tissue which traverse it. The tint is white or greyish-pink, reddish-brown or dark-brown, according to the amount of blood present. The latter tint prevails where there are cavernous blood-vessels with haemorrhagic infiltrations. The consistence of the tumour varies with the proportion of cells it contains, the round-celled form being the softest. The acini surrounded by neoplastic tissue often survive for a long time. Sarcomatous tumours have been described by Wölfler in which muscle-fibres appeared to be included.

Secondary growths are set up in consequence of invasion of the lymphatics or blood-vessels.

Wölfler describes a case of **fibroma** in a man aged fifty-six: it took the form of multiple hard nodes of about the size of a walnut.

Acute inflammation of a normal or goitrous gland (acute thyroiditis or *strumitis*) occurs as a result of traumatic injury, and of septic or pyaemic infection, also after typhoid fever, diphtheria (Brieger), tonsillitis, and articular rheumatism; it may also arise idiopathically and cause more or less painful swelling of the part. The inflammation of the gland may thus be due to various kinds of infective microbes. If suppuration takes place one or more pus-cavities or abscesses or even patches of gangrene result, and these sometimes rupture into the surrounding parts.

Chronic inflammation and induration are usually associated with necrosis and disintegration within the substance of goitrous glands. Any other form of chronic inflammation leading to diffuse induration must be extremely rare.

Tuberculosis of the thyroid gland is not very common, though in the disseminated haematogenous miliary affection eruptions of tubercle are met with in it. Larger tuberculous nodes have also been described.

Gummata of the thyroid are very rarely met with.

Hydatids due to *Echinococcus* are also infrequent.

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CHAPTER XC

THE THYMUS GLAND

277. The **thymus** is a gland-like body which grows to a considerable size in the foetus and during the first two years of infancy; after that however it ceases to grow, and at about the tenth year undergoes retrograde change into fibrous and adipose tissue.

It lies in the superior mediastinum behind the first piece of the sternum, extends upwards nearly to the thyroid, and is made up chiefly of two flat elongated lobes which are in contact or coherent along their medial borders and are enclosed in thin connective tissue. The lobes are subdivided into lobules by fibrous septa, and these into structural units or acini, which are more or less completely separated from each other by connective tissue. The acini closely resemble lymph-glands, and are composed of a loose reticular or adenoid stroma filled with indifferent or lymphoid elements and larger multinuclear cells. In the peripheral parts of the acinus the stroma is somewhat closer and more densely interspersed with cells than in the centre, and thus even in the smallest acini a cortical and a medullary layer can be distinguished. The thymus possesses no duct, but has numerous lymphatics whose exact course is however only imperfectly determined. Its tissue has an abundant blood-supply.

Small **accessory glands** are not uncommon; they usually lie above the gland and near the thyroid. Congenital absence of the gland is usually observed only in highly-malformed foetuses; it is very seldom wanting in infants that are otherwise normal.

The weight of the thymus in the new-born infant is about 24 grammes, in a child of two it is about 26 grammes; but this weight is subject to considerable variation in different cases.

According to STIEDA, KÖLLIKER, HIS, and WATNEY, the thymus developes from the epithelium of a branchial cleft and is thus originally an epiblastic structure. The epithelial cells however disappear after a time, and the development of the characteristic lymphadenoid tissue starts from mesoblastic (connective-tissue) elements.

The **function** and exact significance of the thymus are not certainly known. WATNEY, who has made its structure the subject of comprehensive investigation, thinks that it takes part in the formation of red and white blood-cells. The former are supposed to be developed in certain nucleated cells containing haemoglobin.

Before birth, and in larger number during infancy, the thymus contains homogeneous or indistinctly-laminated partially-calcified bodies known as Hassal's concentric corpuscles. They lie chiefly in the centre of the acini, and are composed of cells closely applied to each other like the coats of an onion.

The laminated bodies, calcified and uncalcified, break down and disappear during the retrogression of the gland, which is manifested chiefly by the dwindling and disappearance of its cells.

Of **morbid changes** in the thymus the commonest are abnormally large size in the early years of life, and imperfect retrogression after the tenth year, by which it sometimes is allowed to persist until the thirtieth or fortieth year.

Haemorrhage into the gland is met with chiefly in asphyxia, or in connexion with the haemorrhagic diathesis and with scurvy.

According to Chiari the gland-tissue in new-born infants sometimes grows into the interior of the concentric bodies, and then breaking down gives rise to cysts containing pus-like matter. These have been erroneously regarded as little abscesses (Dubois), and were at one time thought to be characteristic of congenital syphilis.

Haematogenous purulent inflammation is usually due to pyaemia, and may lead to multiple abscesses or to general suppuration. According to Ribbert and Eberle pus may accumulate in the spaces vacated by the primitive epithelial structures from which the gland originates.

Tuberculosis appears in the form of disseminated nodules and of large caseous granulomatous nodes.

Gummatous inflammatory changes due to syphilis have been several times described.

Primary tumours having the structure of soft or hard lymphosarcoma or of simple sarcoma occur as soft and marrowy or firm growths, and at times reach a considerable size. They may compress the air-passages and blood-vessels, or press upon the heart and displace the lungs laterally.

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SECTION XII

THE URINARY ORGANS

CHAPTER XCI

MALFORMATIONS OF THE URINARY ORGANS

278. The congenital **anomalies** of the urinary organs consist chiefly in variations of form, displacements, and duplication of certain parts. Textural variations in the structure of the kidney sometimes form the starting-point of enormous congenital tumours.

Total **absence** or grave **hypoplasia** of both kidneys occurs only in highly-malformed foetuses, and is of course incompatible with independent life.

Absence of one kidney is rare in foetuses otherwise well developed. It does not interfere with growth and development, inasmuch as the other kidney becomes hypertrophied and assumes the whole work of excretion. The left kidney is more often wanting than the right. The corresponding ureter is usually absent; though in some instances rudiments of the lower extremity of the canal are present.

Congenital hypoplasia of one kidney is more common than entire absence. In well-marked cases the stunted kidney appears as a thin plate of fibrous tissue 2 to 5 centimetres in length and 1.5 to 3 centimetres broad, with few or no traces of tubules or glomeruli, and supplied by renal vessels normal in position but abnormally small.

The causes of unilateral agenesis of the kidney are commonly undiscoverable. We can only say that for some reason the cellular outgrowths from the primitive ureter (mesonephric duct) out of which the permanent kidney is fashioned have been checked in development or altogether suppressed. Hypoplasia of the kidney depends in some cases on a similar arrest of development, of whose precise nature we are equally ignorant; in other cases however it is due to inflammatory changes analogous to those observed in extra-uterine life.

Congenital **hypertrophy** of the kidney occurs in all cases in which one of the organs is absent or stunted, the single organ increasing in size, even after birth, until its weight becomes equal to that of two normal kidneys.

Among congenital **anomalies of form** the persistence of the foetal lobulations is the most common. The boundaries of the primitive renal segments are however usually indicated only by shallow furrows ; it is very uncommon to find the furrows so deep that the segments are entirely separated and abstricted into distinct *renculi*.

Cohesion of the two kidneys most frequently takes the form of the so-called **horse-shoe kidney** (Fig. 452), in which the organs (*a*) are found closer to each other than is normal, and their lower ends are united by a fibrous band or by ordinary renal tissue (*b*). Cohesion of the upper or middle parts, or of the whole of the mesial borders, is very much rarer. More intimate fusion of the two kidneys into one is usually associated with considerable misplacement or **dystopia**. The single organ is often seated near the promontory of the sacrum in the form of a thick cake or disc, from the anterior aspect of which arises a single or double pelvis with from one to four short ureters. In a few cases the united kidneys have been found on one or other side of the spinal column.

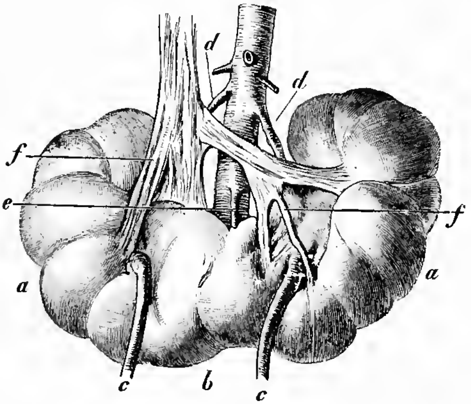

FIG. 452. HORSE-SHOE KIDNEY FROM A NEW-BORN INFANT.

(*Natural size*)

a united kidneys
b connecting piece
c ureters
d renal arteries
e supernumerary renal arteries
f renal veins

The blood-vessels of the united and misplaced kidneys are always abnormal in their origin, and are frequently multiple. Thus when the organ is just above the sacrum the arteries spring from the lower part of the aorta or from its point of bifurcation, and from one of the common iliacs, while the veins enter the corresponding parts of the vena cava or iliac veins.

Unilateral dystopia is most frequent in the case of the left kidney, which approaches the middle line and is displaced downwards to the neighbourhood of the sacrum. The vessels are then abnormal in their origin, and the ureter is shortened.

Acquired **displacement** of the kidneys is commonest on the right side, and is permitted partly by the loose or extensible condition of the perinephral structures, and especially of the peritoneum, which sometimes forms a fold or mesonephron investing the kidney and loosely attaching it to the spine. The vessels are of normal origin and the ureter is not shortened, though it may be twisted or bent on itself. The kidney is moreover in general readily movable, and hence is called **floating kidney**.

On congenital cysts and tumours see Arts. 296 and 297.

Of **malformations of the ureter** and pelvis of the kidney, unilateral or bilateral duplication of the pelvis and ureter is the most usual. It is very rare for the pelvis to be further subdivided into a larger number of tube-like calices.

The duplication of the ureter is either restricted to the upper portion or else it extends throughout the entire course, the two tubes opening separately in the bladder; in the latter case they usually cross each other.

Both normal and abnormal ureters may open in abnormal situations. In the male one of the ureters may open into the colliculus seminalis or into a seminal vesicle; in the female into the urethra, vagina, or uterus. A secondary coalescence of one ureter with a müllerian duct is sometimes observed.

In rare instances valvular folds of mucous membrane which act as valves, and congenital twists or kinks, are found in the tube. Congenital atresia of one ureter or pelvis, or of a single calix, has also been observed. Any of these anomalies may so hinder the outflow of urine as to cause hydronephrosis (Art. 298). According to Boström, Tangl, and others, occlusion of the vesical opening of the ureter sometimes gives rise to dilatation of its lower end into a cyst, which by protruding into the bladder obstructs the opening of the other ureter, or should the end of the blind ureter descend to the base of the bladder it may block the urethral opening.

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279. Of the **malformations of the bladder** the most serious is extroversion (otherwise *ecstrophia*, *extroversio*, *inversio*, or *fissura vesicae*). This malformation is due to the imperfect closure of the abdominal walls and of the bladder, a defect remaining above the symphysis through which the posterior wall of the bladder protrudes. The symphysis in many cases remains likewise unclosed, while the penis or clitoris is rudimentary, and the urethra opens on its upper surface (epispadias).

More rarely the bladder itself is closed and protrudes through the abdominal fissure or through the umbilicus (*ectopia vesicae*). Sometimes the anterior wall is closed while the posterior remains open, a communication existing between the bladder and the pelvic cavity or the vagina.

Very frequently remains of the urachus are found in the lower part of the median vesical ligament, taking the form either of a narrow tube lined with epithelium or of small detached cysts, which may be either closed or open towards the bladder. In the latter case they sometimes become distended with urine when the bladder is overfilled. If any impediment to the outflow of urine takes place in infancy, the urachus may never close at all; and it has been known occasionally to serve as a means of emptying the bladder.

Division of the bladder into two separate (*vesica bipartita*) or partly separate portions (*vesica bilocularis*) is very rare; the two cavities lie side by side or one above another. In the latter case the ureters may enter either the upper or the lower cavity.

Congenital diverticula of the bladder are rare.

Atresia of the vesical orifice of the urethra or of the ureters is also rare. In cases of closure of the urethral opening the urachus usually remains patent. When the opening is obstructed the bladder sometimes becomes enormously distended.

Entire absence of the bladder unaccompanied by any other grave malformation is seldom observed; but the organ is more frequently found to be abnormally small. When the bladder is absent the ureters open into the urethra.

Absence of the urethra is occasionally observed in both sexes.

Atresia of the urethra also occurs in both sexes, being due either to non-development of some part of the canal or to occlusion of its orifice.

The canal may be abnormally narrow either throughout or at some particular part. The contraction is in some cases due to hypertrophy of the colliculus seminalis.

When the urethra opens on the upper aspect of the penis the condition is called **epispadias** (Fig. 453), when it opens on the under aspect **hypospadias** (Fig. 454). The latter is more common: the orifice may be in the neighbourhood of the glans, in the penile portion, or in the anterior or even the posterior attachment of the scrotum (*hypospadias perineoscrotalis*). The penis is usually ill-developed. Occasionally we meet with cases in which the urethra has more than one external orifice, and in males the glans penis is sometimes pierced with what seems to be a second meatus; it is however merely a blind passage ending within the glans.

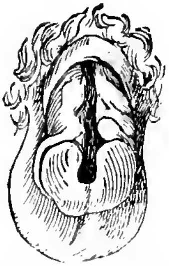

FIG. 453. EPISPADIAS.
(*After* AHLFELD)

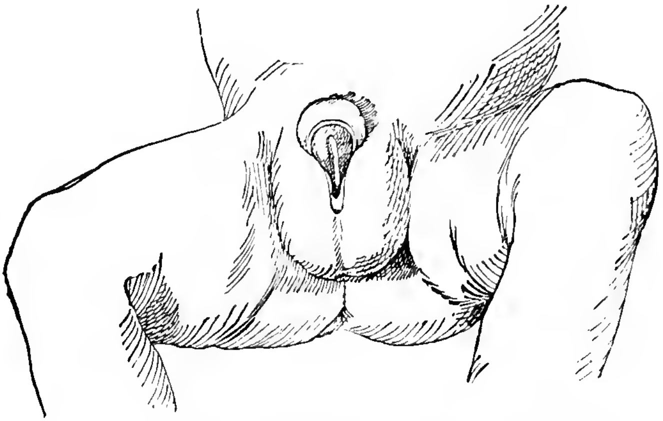

FIG. 454. HYPOSPADIAS WITH ILL-DEVELOPED PENIS.
(*Three-fourths of the natural size*)

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CHAPTER XCII

DISORDERS OF THE RENAL CIRCULATION

280. **Active hyperaemia** or congestion of the kidneys is due either to increased pressure within the aorta, or to dilatation of the renal arteries.

As the secretion of urine is in the main determined by the pressure and velocity of the blood-current through the glomeruli, congestion of the kidneys is accompanied by an increase of secretion.

Passive hyperaemia or engorgement of the kidney is usually the result of some general disturbance of the circulation: it is much less often due to local causes. Affections of the heart and lungs give rise to the former, thrombosis of the vena cava or of the renal veins to the latter. The latter condition occurs most frequently in infants a few weeks old who die of general marasmus; but it also supervenes as a result of inflammatory or other forms of renal disease that are associated with partial obliteration of the renal vessels.

If the outflow of blood from the kidneys is suddenly stopped, they become engorged and greatly swollen, assuming a dark-brown or purple hue. Very soon haemorrhages make their appearance, not only in the cortex and beneath the capsule but also in the medulla, Bowman's capsules and the urinary tubules becoming distended with blood.

If the obstruction of the renal veins is gradual, the blood in part finds its way out through certain small vessels that pass from the kidney into the capsule and empty themselves into venules communicating with the phrenic, lumbar, and suprarenal veins. Accordingly the kidney becomes merely oedematous, few red corpuscles escaping from the engorged blood-vessels.

If however the obstruction is great and persistent the renal tissues become fatty and necrotic, and presently disintegrate entirely.

When the engorgement is less extreme, as in cases of uncompensated cardiac lesion, the swelling of the kidney is but slight, but its colour becomes dark bluish-red or cyanotic. If this condition persists for any length of time, the kidney becomes remarkably dense and firm; at the same time the cortex becomes pale or greyish-red, with darker streaks corresponding to the course of the veins. This change is described as **cyanotic induration**.

When the engorgement is still recent the vessels are uniformly distended with blood, the veins and capillaries being often greatly dilated. Within the capsules of many glomeruli and in the lumen of many of the urinary tubules appears a quantity of liquid, which on boiling yields a granular precipitate of albumen and often contains a few red blood-corpuscles. In some of the tubules lie colourless transparent casts, the so-called **hyaline tube-casts** or cylinders. These are simply masses of albumen which have escaped in liquid form with the watery transudation from the glomeruli, and have become solid within the tubules. Moreover some of the renal epithelial cells, chiefly those of Henle's loops, contain brown or yellow and occasionally crystalline pigment-granules, derived from the colouring matter of the blood-corpuscles that have escaped into the tubules and there become dissolved. If a considerable number of blood-corpuscles have escaped from a glomerulus shortly before the examination is made, the corresponding capsule and the tubule leading from it are often found to be crammed with red corpuscles or the products of their disintegration.

In cases of long-standing engorgement where the kidney is indurated, the intertubular connective tissue is somewhat increased in amount, the blood-vessels are wide and flaccid, and the walls of the capillaries and the adventitia of the veins are thickened. At times some slight degree of inflammatory infiltration with leucocytes is also apparent.

Many of the epithelial cells of the tubules are fatty and contain oil-globules of various sizes. The cells of the straight tubules of the medulla are especially liable to fatty change. The glomeruli appear for the most part unaltered; though here and there a glomerulus is seen whose contents have become homogeneous and shrunken, while the corresponding tubule is narrow, collapsed, or altogether atrophied (Art. 282).

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281. The renal arteries having no arterial anastomoses, when any one of them or of their branches is blocked by an embolus the result is **ischaemic necrosis** of the tissue it supplies.

Immediately after the stoppage of the circulation through it the affected part appears unchanged; but in a few hours the starved tissue dies and gradually assumes a turbid pale-grey or yellowish-white clayey tint, while in the zone immediately surrounding it a certain amount of hyperaemia and haemorrhagic infiltration make their appearance.

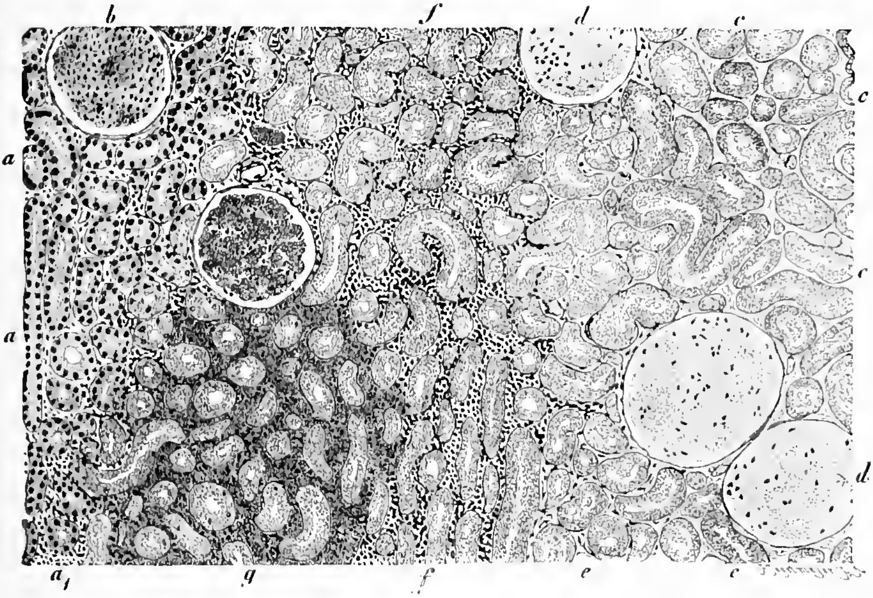

FIG. 455. MARGIN OF AN ANAEMIC INFARCT OF THE KIDNEY.

(Preparation hardened in Müller's fluid, stained with haematoxylin and eosin, and mounted in Canada balsam: × 50)

a normal urinary tubule in normal interstitial tissue
a_1 normal tubule in interstitial tissue infiltrated with round-cells
b normal glomerulus
c necrotic denucleated tissue with granular coagula in the tubules
d necrotic and swollen glomerulus with scanty nuclei
e denucleated tubules surrounded by nucleated interstitial tissue
f cellular and *g* haemorrhagic infiltration of necrotic tissue

The haemorrhage is usually very slight and limited to the marginal zone of the ischaemic region: it very rarely if ever extends over the whole of the part supplied by the occluded artery. The ordinary result of the embolism is therefore the production of an **anaemic infarct** surrounded by a reddened marginal zone.

Within the first few days after the embolism a renal infarct thus appears as a truncated wedge-shaped patch of a dull opaque yellowish-white or greyish-yellow colour, containing dead and denucleated tissue (Fig. 455 *c d*). The tissue surrounding it is somewhat hyperaemic and in parts haemorrhagic (*g*), or infiltrated with round-cells (*f*), so that a red and a greyish marginal zone may sometimes be distinguished round the wedge, especially about its truncated apex.

The smallest infarcts are about the size of a pea; more commonly they are larger, measuring from 4 to 10 millimetres at the base and extending from the surface to the middle of the cortex or even to the boundary between the cortex and medulla. Now and then they are so large as to include a third or even the whole of the kidney, but it should be noted that in large anaemic infarcts and even in anaemic necrosis of the entire kidney small islands of healthy tissue usually persist in the outer strata of the cortex, which receive their blood-supply from the vessels of the capsule.

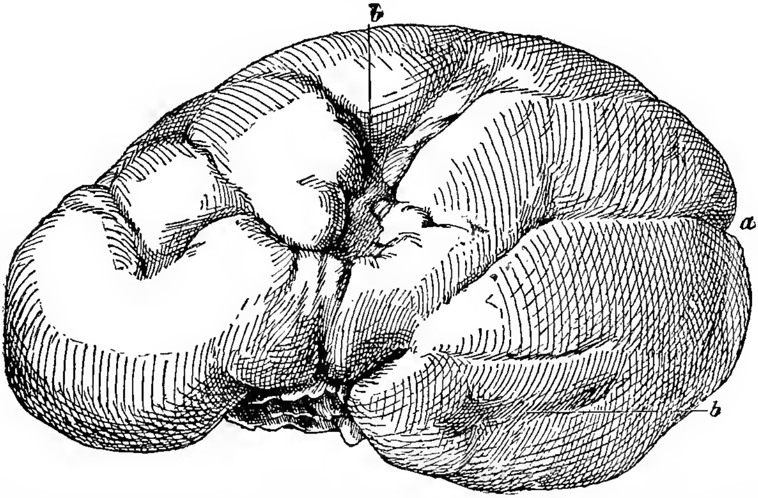

FIG. 456. KIDNEY WITH CONGENITAL FURROWS (*a*) AND EMBOLIC CICATRICES (*b*). (*Reduced to three-fourths of the natural size*)

The tissue that has undergone ischaemic necrosis after some time breaks down and liquefies, and is then re-absorbed, while proliferous changes make their appearance in the parts around. After weeks or months a more or less deeply retracted **cicatrix** (Fig. 456 *b*) is found to occupy the site of the infarct. The size of the cicatrix varies with the extent of the antecedent necrosis; it may be limited to the external layers of the cortex, or it may traverse its whole thickness and even involve the papillae of the medulla. The scar consists essentially of fibrous tissue enclosing hyaline nodules representing the remains of the necrotic glomeruli (Fig. 457 *a*); it has a white, greyish-white, or reddish colour, and at times is spotted with black or brown pigment. The centre of the cicatrix shows no trace of renal tubules, but in the marginal zone surrounding the indurated tissue a few small tubules can be made out (*b*); these are probably atrophic and functionless, but some of them may perhaps be newly formed.

The regenerative hyperplasia that takes place in the neighbourhood of the necrotic patch produces both connective tissue and epithelial cells; but once the glomeruli and tubules have been entirely deprived of blood they are not restored, even though circulation is promptly re-established by the afflux of blood through the adjacent capillaries or through anastomoses with the capsular vessels, or by the re-opening of the embolised vessels as the embolus shrinks. The result is that embolic infarcts invariably leave depressed scars behind them (Fig. 456 *b*).

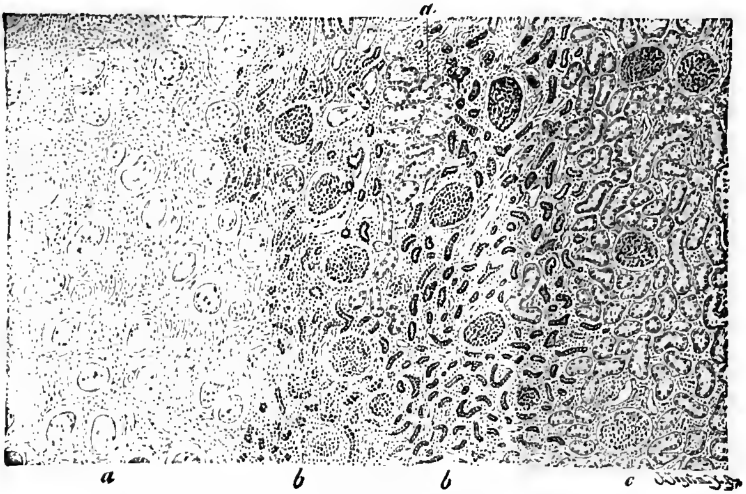

FIG. 457. MARGIN OF AN EMBOLIC CICATRIX.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin*: × 30)

a cicatrix devoid of tubules and containing structureless remains of glomeruli
b indurated tissue with atrophic tubules and glomeruli whose structure is retained
c normal cortical tissue
d normal tubules enclosed in the cicatrix

The number and magnitude of the embolic cicatrices determine the degree of deformation which the kidney as a whole undergoes by their contraction. When they are numerous and large the bulk of the organ is considerably diminished, and a peculiar form of contracted kidney which we may appropriately call the **embolic contracted kidney** is produced.

References on Embolic Infarction of the Kidney.

Argutinski: *Beiträge zur normalen und patholog. Anatomie der Niere* Halle **1877**
Cohnheim: *Allgemeine Pathologie* I Berlin **1882** (trans. by McKee New Syd. Soc.) London **1888**
Foà: Infarcts of the kidney *Ziegler's Beiträge* V **1889**
Frühwald: Haemorrhagic infarcts in both kidneys after cholera infantum *Jahrb. f. Kinderheilk.* new series XXIII **1885**
Guillebeau: *Ueber die Aetiologie der hämorrhagischen Infarkte* Berne **1880**
Hamilton: *Liverpool Med.-chir. Journ.* III **1883**
Israel: Anaemic necrosis of the renal epithelium *V. A.* 123 **1891**
Litten: *Untersuch. über den hämorrhagischen Infarkt* Berlin **1879**
Mögling: *Zur Entstehung d. haemorr. Infarcts* Jena **1884** (with references)
Talma: *Z. f. klin. Med.* II **1884**
Weigert: Morbid coagulative processes *V. A.* 79 **1880**

282. In general **anaemia**, and in spastic conditions of the arteries (as in epilepsy, tetanus, or lead-poisoning), the blood-supply of the kidney is diminished, and it becomes anaemic. This condition is manifested by more or less pronounced pallor of the organ, which assumes a greyish-pink and ultimately a greyish-white tint. Persistent anaemia generally results in fatty degeneration of the renal epithelium.

When from old age or other condition producing arteriosclerosis the walls of the renal arterioles, and in particular the interlobular and afferent vessels, or the glomerular capillaries, are thickened and their channels obstructed or occluded, progressive atrophy of the parts functionally dependent on or supplied by these vessels is induced. The atrophy is proportionate to the extent and degree of the vascular obstruction, and may be described as **senile** or **arteriosclerotic atrophy**, as the case may be.

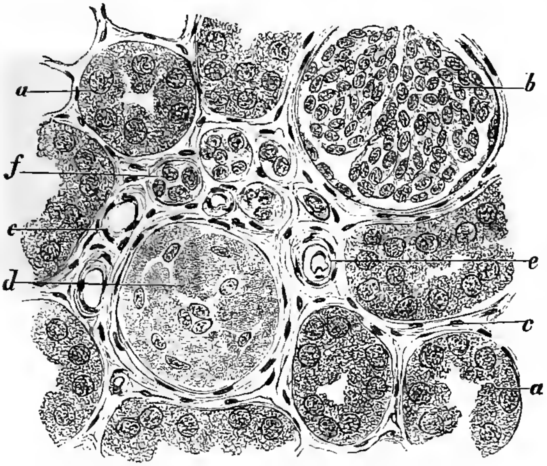

Fig. 458. Senile atrophy of the kidney.
(*Preparation hardened in alcohol, and stained with alum-carmine:* × 200)

a normal tubule
b normal glomerulus
c vascular stroma
d atrophied and functionless glomerulus
e arteriole with somewhat thickened intima
f atrophied and collapsed tubules

When the process of vascular constriction and obliteration begins in the glomerular vessels and *vasa afferentia*, the glomeruli thereby affected are gradually converted into denucleated solid spherules (Fig. 458 *d*), as the glomerular epithelium covering the blood-vessels (*b*), the nucleated endothelium that lines them, and finally the capsular epithelium degenerate and disappear. The capillaries themselves are so altered by hyaline thickening of their walls and obliteration of their channels that they are no longer recognisable as vessels, and the whole glomerular plexus seems made up of homogeneous and structureless segments.

Thickening may take place in the capsule surrounding the shrunken glomerulus, but it is usually inconsiderable.

The urinary tubules corresponding to the atrophied glomeruli always become atrophied also (*f*). They diminish in calibre, and dwindle into slender canaliculi lined with small epithelial cells that stain deeply with the ordinary nuclear reagents.

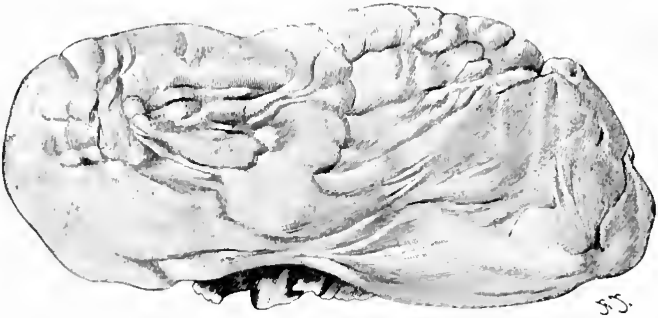

FIG. 459. ARTERIOSCLEROTIC CONTRACTED KIDNEY.
(*Natural size*)

When the obliteration of the vessels is limited to a few isolated glomeruli, no striking change in the section of the kidney is apparent to the naked eye; but the atrophic spots can often be recognised as small isolated cicatricial depressions, and careful inspection reveals that in senile kidneys such atrophic depressions are not infrequent.

The changes are much more pronounced when the renal blood-vessels are the seat of progressive arteriosclerosis. Here the cicatricial depressions are no longer isolated but numerous, and stud the surface of the kidneys so thickly that it looks granular and uneven (Fig. 459), while its bulk is very considerably diminished by the loss of the greater part of its cortical stratum. The resulting condition gives rise to what is aptly called the **arteriosclerotic contracted kidney**.

The cause of this excessive contraction is to be found in the fact that, in this case, not merely a few glomeruli but the greater number of them (Fig. 460 *b c d*) are atrophied and impermeable either by the blood or by injections (Fig. 460), and that the larger interlobular arteries (Fig. 460 *e* and Fig. 461 *a*) are obstructed by thickening of the intima, so that even the glomeruli that may still be permeable have their blood-supply diminished or arrested.

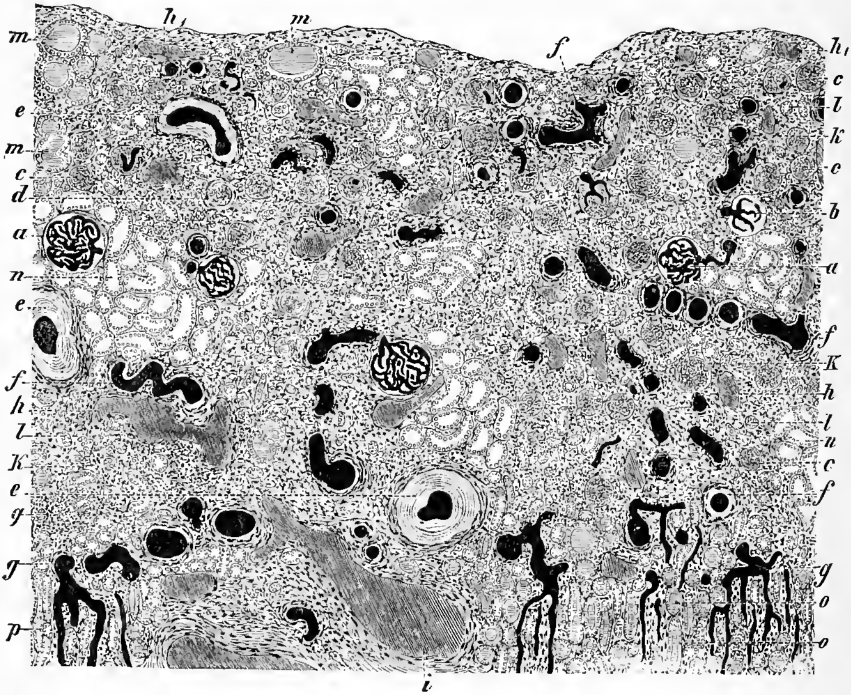

FIG. 460. CORTEX OF AN ARTERIOSCLEROTIC CONTRACTED KIDNEY.

(*Arteries and glomeruli injected with Prussian blue: preparation stained with alum-carmine:* × 50)

a	normal glomeruli
b	partially and *c* totally atrophied glomeruli without thickening of the capsule
d	atrophied glomerulus with thickened capsule
e	artery with greatly-thickened intima
f	interlobular arteries much convoluted and running almost parallel to the surface of the kidney
g	dilated arteries passing down to the medullary zone
h h_1	interlobular and subcapsular veins
i	large venous trunk
k	atrophied parenchyma with a few shrunken tubules *l*
m	cystic dilatation of a tubule with hyaline contents
n	normal tubules
o	tubules in the medullary ray with hyaline casts
p	patent tubules in the medullary ray
q	cellular infiltration

The wasting of the urinary tubules (Fig. 461 *d e*) proceeds *pari passu* with the obliteration of the glomeruli, and thus in the end all the tubules within a particular region (Fig. 460 *k*) become collapsed and shrunken, and contain only small epithelial cells or it may be none at all.

The shrinking and warping of the cortex cause the interlobular arteries that run perpendicularly to the surface to be distorted and twisted into irregular spirals (Fig. 460 *f*). The blood, no longer able to pass through the cortex, is forced in increased quantity into the *arteriae rectae* running to the medulla (Fig. 460 *g*), and they thereby become widely dilated.

The connective tissue of the atrophic region is generally but little affected: in consequence of the general contraction it appears to be more abundant and more abundantly nucleated (*k*) than is normal, and here and there it sometimes does in fact contain an increased number of cells (*q*).

In the channels of the functionless tubules we frequently find homogeneous **colloid cylinders** and spherules (Fig. 460 *m o* and Fig. 461 *f g h*), which usually fill the tubules, or when excessive in size dilate them into little cysts (Fig. 460 *m*) with yellowish homogeneous contents. In rare cases the colloid matter appears within the cysts in the form of a number of separate stratified spherules (Fig. 461 *h*). So far as can be made out with the microscope, these homogeneous masses are a colloid product of the renal epithelium, which is produced either in the form of droplets that afterwards unite, or by a transformation of the entire cell when it is loosened from its point of attachment and carried to a lower portion of the tubule (*g*). The dissolved albumen which flows through the diseased glomeruli may have something to do with the formation of these homogeneous masses (Art. 287).

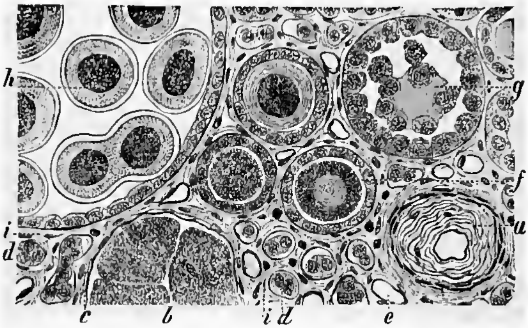

FIG. 461. CONTRACTED KIDNEY WITH ARTERIAL SCLEROSIS.

(*Preparation stained with alum-carmine, and mounted in Canada balsam*: × 150)

a	artery with thickened and fibrous intima
b	obliterated glomerulus denuded of epithelium
c	capsule collapsed but not thickened
d	tubule collapsed and filled with small cells
e	tubule empty and collapsed
f	tubules with stratified and unstratified colloid casts and spherules
g	dilated tubule containing a homogeneous mass beset with shed epithelium
h	cyst containing stratified colloid spherules
i	stroma of cells and delicate fibres

The arteriosclerotic contracted kidney is usually bright-red or greyish-red; but the supervention of fatty degeneration sometimes mottles it with greyish-white or white spots. The contraction in uncomplicated cases is very slow in its progress. The amount of albumen in the urine is slight, and it may at times be absent altogether. In its simpler form the affection is primarily a vascular disease, and may be caused by various kinds of injurious influences (such as lead-poisoning). It must however be remembered that progressive obliteration of the vessels, with thickening of the walls of the larger arteries, may also be a concomitant of chronic indurative or interstitial nephritis; and accordingly the latter affection cannot be sharply distinguished from arteriosclerotic contraction (Art. 294).

References on Arteriosclerotic Contraction of the Kidney (see also Art. 294).

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Hoffa: Nephritis saturnina *Inaug. Diss.* Freiburg **1883**
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Lemcke: Chronic interstitial nephritis and endarteritis obliterans *D. A. f. klin. Med.* XXXV **1885**
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Leyden: Contraction and cirrhosis of the kidneys *D. med. Woch.* VI **1880**, and *Z. f. klin. Med.* II **1880**; Morbid anatomy of lead-nephritis *Z. f. klin. Med.* VII **1883**
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Saundby: Histology of granular kidney *Trans. Path. Soc.* XXXI London **1879**, and *Journ. of Anat.* XV **1880**
Thoma: Disturbances of circulation in the kidneys in chronic interstitial nephritis *V. A.* 71 **1877**
Traube: Lead-poisoning *Allg. med. Central-Ztg.* **1861**
Ziegler: Aetiology of contraction of the kidney with remarks on the differentiation of the various forms of nephritis *D. A. f. klin. Med.* XXV **1880**

CHAPTER XCIII

REGENERATION AND HYPERTROPHY OF RENAL TISSUE

283. When a portion of the **renal epithelium** has been destroyed by a morbid process which spares the interstitial structures, the loss is in general soon made good by regenerative proliferation of the remainder; and if the circulation is adequately maintained the new epithelium presently becomes capable of carrying on the secretory function. But if the lesion is such as to damage irretrievably the renal blood-vessels, regeneration of the epithelium is either wanting, as in the case of the glomeruli (Arts. 281 and 282), or imperfect and partial, as in the urinary tubules; in the latter case the new epithelium never becomes functionally active and remains small and ill-developed.

Traumatic injury of the kidney is repaired by granulations that spring up within the wound, and afterwards develope into a cicatrix, in the manner already described under ischaemic infarction (Art. 281). The cicatricial tissue contains at most a few imperfect tubules, or it may be none at all. So far as can be made out by histological examination, the damaged renal epithelium undergoes a certain amount of proliferation during the process of repair, and new tubules may thus be produced; but these never become functionally active. The glomeruli are not reproduced.

When a considerable amount of the renal tissue is destroyed by disease or removed by operation the remainder undergoes compensatory **hypertrophy,** given normal conditions of the circulation and of nutrition; and the hypertrophy proceeds until the total amount of tissue becomes nearly equivalent to the original amount. During the foetal period and for a certain time after birth the hypertrophic process consists in the production of new glomeruli and tubules; afterwards the existing glomeruli and tubules are simply enlarged by the multiplication of the glomerular and the tubular epithelial cells.

Compensatory hypertrophy of the kidney is thus a functional overgrowth of particular parts, and its extent is determined by the physiological demands the organ has to meet.

According to THOMA (*Untersuchungen über die Grösse und das Gewicht der Bestandtheile des Körpers* Leipzig **1882**) the weight of the kidneys at birth is 23 grammes; at the age of six months, 44 grammes; at the end of the first year, 62 grammes; at ten years, 165 grammes; at twenty years, 285 grammes; and at twenty-five years, 304 grammes. Even under normal conditions the weight of one adult kidney may differ from that of the other by 30 or 40 grammes.

According to LEICHTENSTERN the diameter of a normal glomerulus is from 135 to 225 microns (micro-millimetres); that of a convoluted tubule, 49 to 79 microns; that of a straight tubule, 26 to 49 microns. In hypertrophic kidneys the first rises as high as from 188 to 402 microns; the second, 49 to 141 microns, and the last, 49 to 89 microns.

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BARTH: Repair of wounds of the kidney *A. f. klin. Chir.* XLV **1892**
BEUMER: Renal injuries *V. A.* 72 **1878**
BOSTRÖM: *Beiträge z. path. Anat. der Niere* Freiburg **1884**
ECKARDT: Compensatory hypertrophy and physiological growth of the kidney *V. A.* 114 **1888** (with numerous references)
FRAENKEL: *D. med. Woch.* XIV **1888**
GOLGI: Compensatory hypertrophy of the kidney *A. per le scienze med.* VI **1882**, and *A. ital. de biol.* II **1882**
GRAWITZ and ISRAEL: Renal disease and cardiac hypertrophy *V. A.* 77 **1879**; Compensatory hypertrophy of the kidney *V. A.* 88 **1882**
GUDDEN: Extirpation of the kidney in rabbits *V. A.* 66 **1876**
KÖSTER: Compensatory hypertrophy in atrophic kidneys *Berl. klin. Woch.* **1882**
LEICHTENSTERN: Compensatory hypertrophy *Berl. klin. Woch.* XVIII **1881**
LORENZ: *Z. f. klin. Med.* X **1886**
MURK: Compensatory hypertrophy of the kidney *V. A.* 111 **1888**
PERL: Idem *V. A.* 56 **1872**
PISENTI: Cicatrisation of wounds of the kidney *A. per le scienze med.* VIII **1884**
PODWYSSOZKI: Regeneration of glandular tissue *Ziegler's Beiträge* II **1887**
RIBBERT: Compensatory hypertrophy of the kidney *V. A.* 88 **1882**
ROSENSTEIN: Complementary hypertrophy *V. A.* 53 **1871**
TILLMANNS: Researches on wounds of the liver and kidney *V. A.* 78 **1879**
TUFFIER: *Études expér. sur la chirurgie du rein* Paris **1889**
ZIEGLER: Causes of morbid proliferation *Virchow's Festschrift* (*internationale*) II Berlin **1891**

CHAPTER XCIV

RENAL DEPOSITS DERIVED FROM THE BLOOD

284. The kidney is not infrequently the seat of deposits of **corpuscular matters**, either brought to it as such or precipitated in the solid form within its substance. Some of these deposits are related to the secretory function of the kidney and represent some constituent of the urine that has not been carried off in the usual way; but other deposits have no connexion with secretion. The deposition is due in some instances to morbid conditions existing within the kidney, in others to conditions external to it.

Blood deposited in the renal tissue may in the first place be extravasated from the interlobular vessels (Fig. 455 *g*), in which case it infiltrates the intertubular connective tissue. The causes of haemorrhagic extravasations of this kind are excessive engorgement, arterial embolism, inflammatory processes, and traumatic injuries. Another form of haemorrhage is that due to bleeding from the glomerular vessels, the blood filling up the capsular space and thence escaping into the corresponding tubules (Fig. 476).

The extravasation of small numbers of red blood-corpuscles from the glomeruli is observed in passive hyperaemia and in certain forms of inflammation: copious glomerular haemorrhage is associated with more intense inflammatory irritation. Some of the blood that escapes into the renal tubules is carried away with the urine, the remainder disintegrates *in situ* into yellowish or brownish granular tube-casts and yellowish pigment-granules enclosed in some of the epithelial cells.

Infiltration of the kidney with **leucocytes**, when it is not due to inflammation, is one of the results of leukaemia; it is often manifested as an excessive accumulation of lymphoid cells in the intertubular (Fig. 462 *b*) and circumglomerular tissue. The kidney becomes pale-grey in colour and is somewhat swollen; and at times the cells may even be aggregated into whitish nodes scattered through the organ.

Deposition of **blood-pigment**, sometimes also called haemoglobin-infarction, takes place when in some part of the circulation haemoglobin passes into and is dissolved in the blood-plasma, or red blood-corpuscles have undergone disintegration within the vessels. The pigment that is thereupon deposited in the kidneys is either **haemoglobin** and methaemoglobin, forming reddish-yellow or brownish globules, or **haemosiderin**, deposited as flakes and granules, or **haematoidin**, which takes the form of granules or crystals. These give rise to yellow, reddish, or brown mottling, or diffuse yellow or brown staining (with methaemoglobin), of the renal tissues.

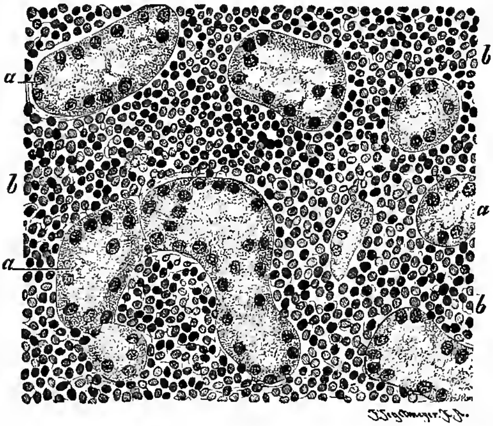

FIG. 462. LEUKAEMIC INFILTRATION OF THE RENAL CORTEX.

(Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin: × 150)

a renal tubules *b* lymphoid cells

The blood-pigment lies either within the vessels or in the fibrous stroma, in the lumen of the tubules, and in Bowman's capsules. Some of it may even be embedded in the tubular epithelial cells. In the epithelium of the convoluted tubules haemosiderin either takes the form of isolated granules or is diffused through the entire protoplasm of each cell, the Prussian-blue test for iron giving a uniform blue tint to the whole cell-body.

The deposition of **bile-pigment**, or biliary infarction, either stains the kidney a uniform yellow or green, or mottles it with these tints. The pigment gives rise to diffuse yellow and green coloration of the epithelium of the convoluted tubules, and appears likewise as a granular deposit in some of the cells (Fig. 463 *a d*). Degeneration and desquamation of these pigmented cells (*c d*) are not uncommon; and bile-stained hyaline casts (*b*), some of them beset with pigmented cells (*c*), are thus formed in the lumen of the tubules. In rare cases the deposits are crystalline.

Pigmentation by **silver** (argyrosis, argyria, or silver-staining) is due to the deposition in the kidney of silver-particles in cases where preparations of the metal have been medicinally administered for a long time. The particles give a smoky-grey or black tint to the renal tissue. This deposition takes place in the fibrous stroma, the silver assuming the form of little brownish-black grains or spherules lying chiefly in the interlobular tissue of the pyramids, but they may also appear in the glomeruli. Copious deposits of silver sometimes cause hyperplasia and induration of the connective tissue, and parts of this undergo calcification.

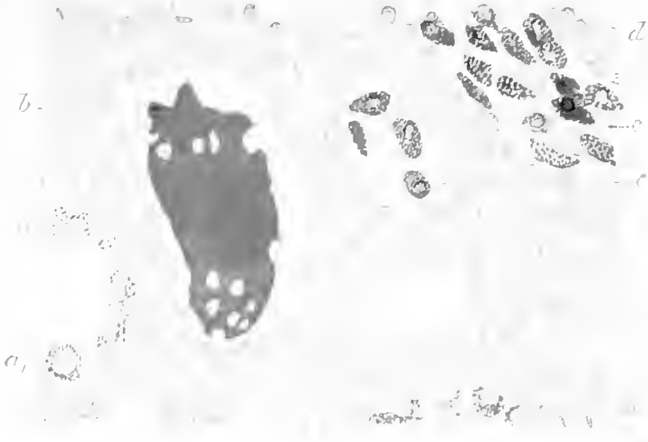

FIG. 463. BILIARY INFARCTION OF THE KIDNEY.

(*Preparation hardened in corrosive sublimate, and stained with carmine:* × 200)

a tubular epithelium with yellowish-green granules
b yellowish-green tube-casts
c cast enclosing pigmented cells
d desquamated epithelium with granules of bile-pigment

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AFANASSIEW: Changes in the kidney in haemoglobinuria *V. A.* 98 **1884**
BOSTRÖM: *Ueber die Intoxicationen durch die essbare Morchel* Leipzig **1882**
FRAENKEL, E.: Morbid changes after death from burns of the skin *D. med. Woch.* **1890**
FROMMANN: Case of argyrosis *V. A.* 17 **1859**
HEIDENHAIN: Excretion of sodium indigo-sulphate *Pflüger's Arch.* 9 **1872**
JAHN: Argyria *Ziegler's Beiträge* XVI **1894**
VON KAHLDEN: Deposit of silver in the kidneys *Ziegler's Beiträge* XV **1894**
LEBEDEFF: Changes of the kidneys from the precipitation of haemoglobin *V. A.* 91 **1883**
MARCHAND: Poisoning by chlorates *V. A.* 77 **1879**, and *A. f. exp. Path.* XXII and XXIII **1887**
PAUTYNSKI: Excretion of sodium indigo-sulphate *V. A.* 79 **1880**
PONFICK: *Berl. klin. Woch.* 17 **1876** and 46 **1877**; *V. A.* 88 **1882**; *Verhandl. der II Congress. inn. Med.* Wiesbaden **1883**
RIEMER: Case of argyria *A. d. Heilk.* XVI **1875** and XVIII **1876**

285. **Uric acid**, whether produced in normal or in abnormal quantity (as in gout and the uric-acid diathesis), may be deposited in the kidney or its pelvis in the solid form or as solid urates when the water excreted is incapable of holding it all in solution, or when certain conditions exist that favour its separation and deposition in these forms.

The deposit consists of amorphous granular masses or of acicular crystals, that are either precipitated in the renal tubules and the pelvis, or incrust organic matters lying in the urinary tract and derived from disintegrated tubular epithelium or from the mucous membrane of the pelvis of the kidney. The granular deposits are known as sand or **gravel**, the incrustations as uratic concretions or **renal calculi.**

In **gout** white lines and streaks are often found in the substance of the renal papillae; they mark the course of deposits within the dilated tubules, which consist of rhombic acicular colourless crystals of acid sodium urate.

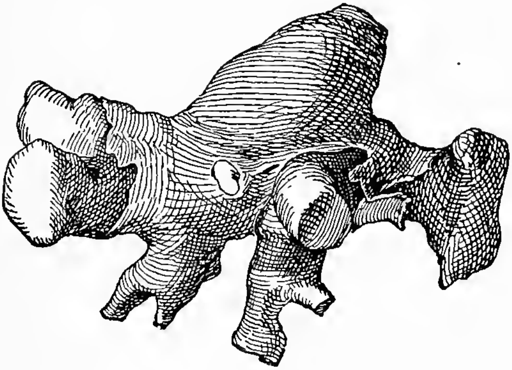

FIG. 464. URATIC CALCULUS FROM THE RENAL PELVIS.
(*Natural size*)

Deposits of urates and uric acid within the kidney are most frequently met with in new-born infants, and give rise to so-called infantile uratic infarction. The granular urates lie in the form of small spherules within the collecting tubules of the medulla, and *en masse* appear as yellowish-red streaks. The infants are in general such as die between two and fourteen days after birth. In infants dying within the first two days of life and in those that have not breathed they are rarely found.

Larger aggregations of urates, described as gravel or renal calculi, form yellowish, reddish, or brownish grains or gritty masses and actual stones, the former lying in the renal tubules or in the pelvis, the latter in the pelvis only. Calculi vary in size from that of a pea to that of a bean or hazel-nut. Occasionally they take the form of large branching casts of the infundibulum and its subdivisions (Fig. 464), and then look something like masses of coral. They arise chiefly in gouty persons, but occur in others also. Inflammation of the renal pelvis favours their production.

References on Uratic Deposits and Renal Calculi.

CHARCOT: *Leçons sur les maladies du foie et des reins* Paris **1882**; *Maladies des vieillards (goutte et rhumatisme) Oeuvres complètes* VII Paris **1890**

EBSTEIN: *Die Natur und die Behandlung der Gicht* Wiesbaden **1882** (trans. by SCOTT) London **1885**; *Die Natur und Behandlung der Harnsteine* Wiesbaden **1884**

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FÜRBRINGER: Nephrolithiasis, renal calculi and concretions *D. med. Woch.* **1890**
GARROD: *On gout and rheumatic gout* London **1876**
HELLER: *Die Harnconcretionen* Vienna **1860**
STRÖBE: Recent researches on the organic matrix of urinary gravel *Cent. f. allgem. Path.* I **1890**
VIRCHOW: *Berl. klin. Woch.* **1884**

References on Uratic Infarction in Infants.

HECKER: *V. A.* 11 **1856**
LANDOUZY: *Bull. Soc. Anat.* XLVII Paris **1872**
LIMAN: *Handb. der gerichtl. Med.* II **1882**
RAPHAEL: *B. M. J.* I **1870**
SCHLOSSBERGER: *A. f. physiol. Heilk.* I **1842** and IX Stuttgart **1850**
SCHULTZE, B.: *Deutsche Klinik* Berlin **1858**
VIRCHOW: *Gesammelte Abhandlungen* Frankfort **1856**

286. Concretions of **calcium phosphate** and **calcium carbonate** deposited in the kidney constitute what is called calcareous infarction. It occurs chiefly in aged persons, in whom resorption of the bony structures is active; but it sometimes makes its appearance when no such resorption is going on, especially when the epithelium of the urinary passages is undergoing destructive changes. According to KAUFMANN, NEUBERGER, PRÉVOST, SALKOWSKY, SENGER, VIRCHOW, and others, calcareous deposits are very apt to be formed in cases of poisoning with corrosive sublimate that are not immediately fatal. According to AFANASSIEW, such deposits also occur in glycerine-poisoning: PALTAUF has met with them in phosphorus-poisoning, and NEUBERGER in poisoning from excessive doses of aloin and bismuth subnitrate. The calcium-salts are precipitated in the convoluted tubules of the cortex, and more rarely in the collecting tubules of the medulla, which have become necrotic (Fig. 465 *c d e*) as a result of ischaemia or of the action of the poison. According to LEUTERT, poisoning with corrosive sublimate, in addition to calcification of the

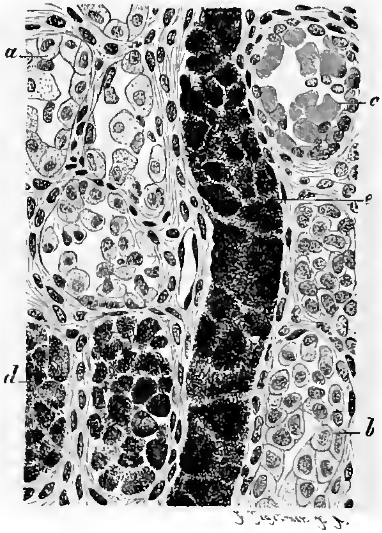

FIG. 465. CALCIFICATION OF THE TUBULAR EPITHELIUM FROM POISONING WITH CORROSIVE SUBLIMATE.

(*The patient died five days after taking the poison: preparation hardened in alcohol, and stained with haematoxylin and eosin:* × 300)

a normal renal tubule
b tubule with desquamated epithelium
c tubule with desquamated and necrotic epithelium containing no nuclei
d e tubules with necrotic and calcified epithelium

necrotic epithelium and of the tube-casts derived from it, gives rise to calcareous deposits in cells that are still active and accordingly retain their nuclei. In connexion with the calcareous metastasis that accompanies increased osseous resorption, calcareous granules sometimes appear in the glomerular capsules and in the indurated intertubular tissue of the medulla. The indurated fibrous tissue that is produced as a result of argyrotic infiltration occasionally becomes calcified.

Slight degrees of calcification can be recognised only with the microscope; more abundant deposits produce a chalky white coloration. In cases of poisoning, the cortex looks more or less turbid, owing to degeneration (*b*) and necrosis (*c*) of the epithelium.

Calculi composed of calcium phosphate or calcium carbonate are white. Calculi of pure chalk are rarely met with; but stones of mixed composition often contain calcium phosphates and carbonate.

Oxalic acid, whether ingested with the food or produced by the decomposition of uric acid, may be deposited in the kidney or its pelvis as 'dumb-bells' or octahedral crystals of calcium oxalate. This occurs when the amount of acid sodium phosphate in the urine is insufficient to maintain in solution the proportion of oxalic acid that is present. Within the kidney the oxalate forms white deposits; in the pelvis it forms pale or dark-brown 'mulberry' or spinous calculi. Calculi of pure calcium oxalate are very rare. The salt more frequently occurs as an incrustation on uratic calculi.

Triple phosphate of ammonium and magnesium occurs as soft crumbly white concretions, seldom pure, but frequently forming a coating on uratic or other calculi. The deposit is produced chiefly in cases of ammoniacal decomposition of the urine; ammonium carbonate is first formed, and this tends to render the urine alkaline and so precipitates the earthy phosphates. The crystals of triple phosphate have usually the so-called 'sarcophagus-form,' derived from a rectangular prism by cutting off the angles and edges.

In rare cases renal concretions and calculi are found which consist of **cystin**, an abnormal constituent of the urine crystallising in hexagonal plates. Cystin-calculi are round, soft, and of a waxy or greenish tint; they have a radiate crystalline structure on fracture. According to BAUMANN, UDRÁNSKY, and BRIEGER, cystin, which is a sulphur-derivative of lactic acid, appears in the urine as a result of a peculiar decomposition of albumen in the intestine, and this decomposition is in turn dependent upon the presence of certain special kinds of bacteria in the alimentary tract.

Xanthin-calculi are extremely rare; they are pale or dark-brown, and not unlike uratic calculi.

All the forms of renal concretions and calculi may give rise to inflammation in one or both kidneys. The condition of a kidney containing concretions in its pelvis is frequently referred to as **nephrolithiasis** (Art. 299).

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Afanassiew: Morbid changes in the kidney and liver in poisoning associated with haemoglobinuria or jaundice *V. A.* 98 **1884**
Ebstein: *Die Natur und Behandlung der Harnsteine* Wiesbaden **1884**
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Leutert: Poisoning with corrosive sublimate *Fortschr. d. Med.* XIII **1895**
Litten: Haemorrhagic infarcts and the influence of arterial anaemia upon living tissue *Z. f. klin. Med.* I **1879**, and *V. A.* 80 **1880**
Neuberger: Action of corrosive sublimate upon the kidneys *Ziegler's Beiträge* VI **1889**; Deposits of calcium salts in the kidneys *A. f. exp. Path.* XXVII **1890**
Paltauf: Phosphorus-poisoning *Wiener klin. Woch.* **1888**
Prévost: Researches on mercurial poisoning *Rev. méd. de la Suisse romande* **1882**
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Virchow: Metastasis of calcium salts *V. A.* 8 **1855**, and 9 **1856**; Poisoning with mercuric cyanide *München. med. Woch.* **1888**

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Ebstein: Cystinuria in articular rheumatism *D. A. f. klin. Med.* XXIII **1878** and XXX **1882**
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Steffenhagen: *V. A.* 100 **1885**

287. When the glomeruli and their epithelium are seriously injured, or the circulation through them greatly disturbed, certain components of the blood which normally are held back are liable to escape from their vessels, and in like manner from the intertubular capillaries, into the renal tubules. This is most notably the case with regard to the **serum-albumen** of the blood, which in morbid conditions passes in greater or less amount into the urine (**albuminuria**).

This albumen comes from the glomeruli in the soluble form; but within the tubules it may coagulate and thus give rise to granular or homogeneous casts, especially in the region of the loops of Henle. These casts are known as **hyaline casts** or cylinders. In many affections of the kidney, especially those of an inflammatory kind, the renal epithelium degenerates or breaks down and desquamates. Moreover we know that from the glomeruli and tubules there escape not only serum-albumen but also white and red blood-corpuscles. In many morbid affections therefore the tubules contain not only soluble albumen but also albumen derived directly from the protoplasm of cells, and this albumen like the other may take part in the formation of tube-casts.

In the first instance the desquamated epithelial cells lie close together and become agglutinated into casts of the tubules; these have received the name of **epithelial casts**. So also the granular albuminoid and fatty products of their disintegration may in like manner give rise to **granular casts**. Again the epithelial cells and leucocytes or their albuminous detritus are sometimes transformed and fused within the tubules into compact hyaline masses, or homogeneous droplets escape directly from the bodies of the degenerating cells and coalesce into translucent cylinders. The agglutination of the products of blood-disintegration in the tubules leads to the formation of **granular blood-casts**.

Tube-casts may in certain circumstances be washed out of the tubules by the urine, and so escape from the kidney. The greater number however remain *in situ*, and are either redissolved or become more firm and dense so as somewhat to resemble wax (**waxy casts**).

In addition to these casts, formed at least in part from transuded albumen, we may have homogeneous cylinders of the renal tubules which are purely epithelial in origin. These have been described (Art. 282, Fig. 461) as **colloid casts**.

References on the Formation of Tube-casts.

Burkart: *Die Harncylinder* Berlin **1874**

Cornil: Epithelial origin of tube-casts *Journ. de l'anat.* XV **1879**, and *Practitioner* XXVIII London **1882**

Ernst: Fibrin in hyaline casts *Ziegler's Beiträge* XIII **1893**

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Knoll: *Prager Z. f. Heilk.* V **1884**

Langhans: Changes in the glomeruli in nephritis with remarks upon the production of fibrinous casts *V. A.* 76 **1879**

Lépine: *Die Fortschritte der Nierenpathologie* Berlin **1884**

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Posner: Albuminous origin *Cent. f. med. Wiss.* **1879** and *V. A.* 79 **1880**

Ribbert: *Nephritis und Albuminurie* Bonn **1881**; Formation of hyaline casts *Cent. f. allg. Path.* IV **1893**

Rosin: *Art.* Tube-casts *Eulenburg's Jahrb.* III **1893**

Rovida: *Moleschott's Untersuchungen* XI Frankfort **1867**

Saundby: *Birmingham Med. Review* **1883** (with references)

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Török and Pollak: Origin of homogeneous tube-casts *A. f. exp. Path.* XXV **1889**

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CHAPTER XCV

HAEMATOGENOUS DEGENERATIONS AND INFLAMMATIONS OF THE KIDNEY

288. The kidneys are glandular organs by means of which a number of products of metabolic waste are removed in aqueous solution from the body. The urinary water is separated from the blood in the glomeruli, and owing to their peculiar structure the liquid that passes from the glomerular vessels into the intracapsular space and the renal tubules contains little or no albumen. The substances dissolved in the urine are eliminated partly in the glomeruli and partly in the tubules, and so must pass through not only the walls of the blood-vessels but also the epithelium investing the glomeruli and tubules.

When the blood receives from without certain abnormal substances which have to be eliminated unchanged or in some new form, or when morbid products are elaborated within the body by perverted metabolism of the food or of the tissues, it is chiefly by the kidneys that they are removed. Some of these substances pass through the kidney without inducing any changes in its structure; but many chemical bodies during the process of elimination exert an injurious influence on the vessel-walls or on the epithelium of the glomeruli and tubules, the injury being manifested by disorders of secretion and by degenerative and inflammatory changes in the renal parenchyma.

The most important indication that the renal function has become disordered is the appearance of albumen in the urine, or **albuminuria**. This is presently accompanied by other symptoms, such as increase or decrease of the quantity of urine daily voided and the presence of other abnormal constituents in the urine. Among the latter are tube-casts (Art. 287), degenerate cells from the tubules, leucocytes, and red blood-corpuscles or the products of their disintegration.

When these symptoms of renal disorder are well marked, the composition and contents of the urine indicating the existence not merely of a transient disturbance of the circulation but of some pathological alteration of the secreting organ, the morbid condition is clinically described as **Bright's disease**, which according to its course and duration is distinguished as acute, subacute, or chronic.

The changes in the parenchyma are primarily of a degenerative character, and affect the vascular connective tissue as well as the epithelial structures. Usually some inflammatory exudation and proliferation are superadded, and result in the reproduction of lost tissue-elements and the hyperplasia of others.

Attempts have often been made, both from the clinical standpoint and from the pathological, to draw sharp distinctions between the purely degenerative and the inflammatory diseases of the kidney; but it is found impracticable to divide them by any clear line. For though the proportion of albumen in the urine has been taken as a clinical criterion whereby to differentiate cases of simple degeneration from cases of nephritis, histological examination of the corresponding kidneys shows that morbid conditions which are essentially degenerative often give rise to graver disorder of the renal secretion than do undoubtedly inflammatory affections. It is therefore better to consider degenerative and inflammatory changes together, and to classify the various forms of Bright's disease by their general course and duration, with the aid of certain outstanding anatomical features of the diseased kidney.

The modern investigations of the affections included under the term nephritis begin with the observation of Bright (*Report of medical cases selected with a view of illustrating the symptoms and cure of diseases by a reference to morbid anatomy* I London **1827**) that certain cases of dropsy depended on disease of the kidney, and were distinguished by albuminous urine. Bright himself described various forms of renal disease leading to albuminuria.

The diseases of the kidney which were first accurately differentiated by Bright have since been included under the term **Bright's disease** (*morbus Brightii*); but the term has been variously applied by different authors — some including under it all renal affections associated with albuminuria, others excluding the simple degenerations and disorders of circulation and including only the inflammatory affections.

Rokitansky (*Handb. d. path. Anat.* II **1842**) distinguished eight forms. Frerichs (*Die Bright'sche Nierenkrankheit* Brunswick **1851**) regarded the different forms merely as different stages of one and the same process. This process, he held, began with hyperaemia, passed on to exudation and parenchymatous degeneration, and ultimately issued in atrophy and contraction.

Klebs excluded the non-inflammatory renal degenerations from the category of Bright's disease, and identified the latter with primary interstitial nephritis: the associated changes in the epithelium he regarded as secondary.

Johnson regarded the presence or absence of epithelial degeneration and desquamation as an essential feature, and divided nephritis into a desquamative and a non-desquamative form, each form having its subordinate varieties.

Grainger Stewart speaks of Bright's diseases, and distinguishes three forms — the inflammatory, the amyloid, and the cirrhotic or contracting. Of the first he describes three stages — that of inflammatory exudation, of fatty change, and of atrophy. Virchow (*Cellular Pathology* London **1860**) also distinguishes three forms — parenchymatous nephritis, indurative interstitial nephritis, and amyloid degeneration. Bartels divides Bright's disease into — acute parenchymatous, chronic parenchymatous, and interstitial nephritis. Lecorché distinguishes only a parenchymatous and an interstitial form. Charcot, on grounds partly clinical and partly anatomical, makes three — the first characterised clinically by its rapid course, scanty urine with abundant albumen, and dropsy, and anatomically by its large white kidney; the second by its chronic course, abundant urine with diminished albumen, absent or slight dropsy, and contracted kidney; the third form is amyloid degeneration. WEIGERT divides Bright's disease into parenchymatous degenerations and true nephritis; the former are all acute affections; the chronic forms are but modifications of one and the same process: he deems it impossible to distinguish interstitial from parenchymatous forms, inasmuch as all forms begin with degeneration and loss of epithelium, and then pass into the stage of reactive interstitial inflammation.

DICKINSON makes three classes — tubal nephritis (acute and chronic), granular degeneration with hyperplasia and contraction of the stroma, and depurative disease (or amyloid degeneration).

AUFRECHT speaks of an acute, a subacute, and a chronic nephritis, and maintains that the primary change is an affection of the tubular epithelium, the vessels and the fibrous structures being affected secondarily: he describes amyloid disease as a nephritis. WAGNER considers that Bright's disease is a clinical term, implying an affection in which the urine exhibits certain morbid changes: he treats it under the four heads of (1) acute Bright's disease, (2) chronic Bright's disease, (3) contracted kidney, (4) amyloid kidney. LEYDEN (*Verhandlungen des Congresses für innere Medicin* Wiesbaden **1882**) defines the term Bright's disease from the clinical or physiological point of view, and comprehends under it all the diseases of the kidney that cause albuminuria and dropsy, including degenerations of the secreting structures, pyelonephritis, amyloid change, etc. ROSENSTEIN divides diffuse inflammation of the kidney, which he makes equivalent to Bright's disease, into acute and chronic diffuse nephritis; and distinguishes in the latter three forms characterised anatomically by the large white kidney, the mottled or smooth contracted kidney, and the granular contracted kidney. CORNIL prefers to use the general term albuminous nephritis, and treats the various forms under the heads of acute nephritis, parenchymatous or epithelial nephritis, and interstitial nephritis. HAMILTON (*Text-book of Pathology* II London **1894**) discusses Bright's disease under the heads of catarrhal nephritis, wax-like kidney, and cirrhotic kidney, each with several subdivisions.

The above summary shows how widely authorities differ as to the content of the term Bright's disease, and as to the morbid anatomy and pathogenesis of nephritis. We might easily carry our references further and so bring out still greater differences. This is true not only of the older authorities, but even of the most recent, the latest discussions on the subject in medical congresses showing clearly that, on the basis of our present knowledge, no reconciliation of the conflicting views is practicable.

The experimental researches on nephritis made by GRAWITZ and ISRAEL, PONFICK, LASSAR, MARCHAND, AUFRECHT, BUCHWALD, LITTEN, and others have but slight bearing on the questions raised by the phenomena of nephritis in man. The varieties of renal degeneration set up by the injection or administration of various chemical irritants, or by the interruption of the blood-supply, etc., admit of useful comparison with the human affections exactly corresponding to them and with no others. Still less have the degenerations of the kidney induced by ligation of a ureter to teach us concerning the textural changes in human haematogenous nephritis. For this latter we must in the first place have recourse to careful anatomical investigation of the diseased human kidney.

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Greenfield: Summary of current pathology *Atlas of pathology* (New Syd. Soc.) II London **1879**

Hortolès: *Étude du processus histologique des néphrites* Paris **1881**

Johnson: *Lectures on Bright's disease* London **1873**

von Kahlden: Acute nephritis *Ziegler's Beiträge* XI **1892** (with references)

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Roberts: *Urinary and renal diseases* London **1885**

Rosenstein: *Pathologie und Therapie der Nierenkrankheiten* Berlin **1894**

Saundby: *Lectures on renal and urinary diseases* Bristol **1896**

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289. **Acute renal degenerations** and **acute haematogenous nephritis**, when they are not due simply to disorder of the circulation (Arts. 280–282), are induced by infective or toxaemic conditions; the primary irritant may be of extraneous origin, or it may be elaborated within the body itself.

Among the extraneous renal irritants are various poisons, such as corrosive sublimate, chromates, phosphorus, arsenic, cantharides, petroleum, potassium chlorate, etc., whose action is fairly well understood; but there are many other substances which when they reach the vessels are capable of inducing degeneration and inflammation of the kidneys.

Nearly all of the infections that give rise to general diseases or to local and metastatic lesions set up degeneration or inflammation of the kidney. Thus renal affections frequently accompany diphtheria, scarlatina, measles, small-pox, septicaemia, pyaemia, croupous pneumonia, relapsing fever, typhoid fever, erysipelas, cholera, acute rheumatism, yellow fever, and even dysentery, tuberculosis, endocarditis, and local suppurations also.

The renal affection in these diseases is probably in most cases referable to the action of toxic substances elaborated by the virus within the body and in part eliminated through the kidneys. But certain non-infective disorders of metabolism, or the resorption and subsequent excretion with the urine of substances normally eliminated through other organs, may in like manner affect the kidney, as in the case of diabetes, gout, jaundice, and haemoglobinuria. Moreover, in infective diseases it must be borne in mind that, in addition to the toxaemic condition, the kidney may be exposed to direct invasion by the specific microbes. As a general rule, the toxic effect is manifested by degeneration of the secreting structures, the microbic invasion by circumscribed inflammatory lesions; but cases occur in which the presence of bacteria sets up cellular degeneration and necrosis, while the action of toxic substances conveyed to the kidneys by the blood sometimes induces not only cellular necrosis but inflammatory exudation also.

Slight affections of the kidneys can generally be recognised during life by the appearance of small quantities of albumen in the urine. In the more severe affections, clinically described as acute nephritis, the quantity of urine is diminished, its specific gravity is high, it contains much albumen, and its colour is dark and in some instances smoky or blood-stained.

The sediment deposited by the urine on standing consists of leucocytes, and when the urine is blood-stained entire red corpuscles, together with hyaline casts, or occasionally granular casts beset with red corpuscles or their detritus, normal epithelial cells from the collecting tubules, and turbid, swollen, and disintegrated cells from the convoluted tubules. Anasarca is usually but not always present, especially in the forms of the affection that are secondary to other diseases.

The usual outcome of the renal affection is recovery, though fatal uraemia may supervene. Only in rare instances does the acute disease pass into chronic indurative nephritis; more rarely still does it end in what is known as chronic parenchymatous nephritis.

The post-mortem appearances presented by the kidney in acute non-suppurative nephritis are of various kinds; and it is indeed impossible from the character of the urine to predict with any certainty the exact condition of the kidney. In many cases it appears to the naked eye unaltered. More frequently however it looks grey, greyish-white, or mottled with greyish and reddish patches, the variations being determined chiefly by the amount of epithelial degeneration and the presence or absence of hyperaemia in the organ. There is generally some enlargement of the kidney, even to as much as twice its natural bulk, and its tissue then seems abnormally moist and soft, as if it were oedematous. While the cortex is often of a pale-grey or greyish-yellow tint, the medulla looks engorged and cyanotic, and so stands out in sharp contrast with the cortex.

The glomeruli are sometimes visible as red or grey spots, in other cases they are indistinguishable. In severe affections the grey and moist but still translucent cortex appears flecked with the greyish or yellowish-white opaque spots that indicate the presence of fatty degeneration, and sometimes with small circumscribed reddish or reddish-brown round or oval haemorrhagic spots due to extravasation into Bowman's capsules and the uriniferous tubules.

In purulent nephritis that has already advanced to the stage of suppuration the parenchyma of the kidney is more or less swollen and cloudy, and is studded here and there with small yellowish rounded or streaky patches of purulent infiltration, often surrounded by a haemorrhagic areola. In cases where the general affection of the kidney is associated with embolic occlusion of some of its blood-vessels, the swollen and discoloured cortex is interrupted in places by opaque yellowish-white or clay-coloured wedges of necrotic tissue (ischaemic infarcts) often surrounded by a hyperaemic or haemorrhagic zone.

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290. The minute textural changes exhibited by the kidney in acute degeneration and inflammation are as various as the naked-eye appearances; they may affect all the component elements of the kidney, or be limited to particular structures. It is a fact of special interest that cases occur in which the glomeruli alone are affected; and this form of the affection, under the name of **glomerulo-nephritis**, has accordingly been distinguished from

those in which the other structures also are markedly altered. On the other hand there are forms in which the glomeruli are slightly, if at all, affected, while the epithelium of the convoluted tubules, for example, is gravely degenerated (Fig. 471). Indeed it is manifest that the glomeruli, the convoluted tubules, the epithelium of the collecting tubules, or the intertubular vessels may be respectively the parts first and chiefly affected, according to the nature of the injurious agent inducing the nephritis.

The presence of abnormal matters in the intracapsular space is often the only indication of morbid change in the glomeruli. In most cases the morbid contents of the capsule consist of an albuminous exudation from the glomerular vessels, whose albumen is apt to be coagulated during the process of hardening the tissue for microscopical examination, when it becomes apparent as a granular (Fig. 466 *g* and Fig. 467 *i*) or more rarely a hyaline deposit. The albumen may also separate out in a granular or a hyaline form during life or after death, appearing sometimes as a delicate investing film, sometimes as a very abundant deposit. Among the other matters that may be found in the intracapsular space, desquamated glomerular epithelium (Fig. 466 *e* e_1 e_2 e_3, Fig. 467 *h* *g*, Fig. 468 *C* *c*, and Fig. 476 *c*) is extremely common: the cells are sometimes well-preserved (Fig. 466 e_3), sometimes necrotic (*e*) and beset with vacuoles (e_2) or actually vesicular (Fig. 467 *h*), sometimes fatty (Fig. 468 *C* *c*) and in process of disintegration (Fig. 467 *g*).

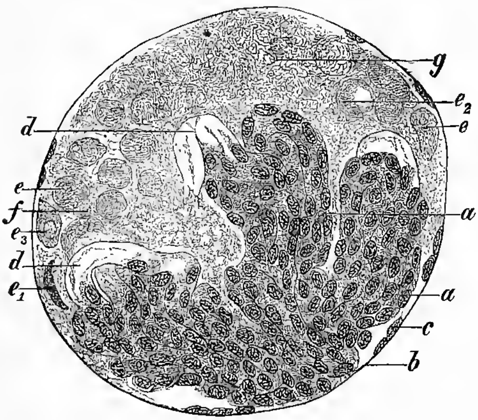

FIG. 466. NECROSIS OF GLOMERULAR EPITHELIUM AND EXUDATION INTO THE CAPSULE OF BOWMAN IN ICTERUS GRAVIS.

(*Preparation hardened in Müller's fluid, stained with gentian-violet, and mounted in Canada balsam:* × 300)

a	normal capillary loop
b	capsule of Bowman
c	capsular epithelium
d	vascular loop denuded of epithelium
e e_1 e_2 e_3	desquamated and degenerate glomerular epithelium
f	exudation between the epithelial cells
g	granular exudation and desquamated epithelium

This appearance is due to the fact that the glomerular epithelium very often degenerates and becomes swollen, fatty, or necrotic; as a result of this the capillary loops are often completely denuded by the exfoliation of their degenerate epithelium (Fig. 466 *d* and Fig. 467 *k*). Usually the capillary epithelium also degenerates, and is thereupon shed in like manner. When acute nephritis persists for some time, continuous desquamation of the new epithelium as it is reproduced may take place, until the intracapsular space is thickly packed with detached cells (Fig. 476 *e g*).

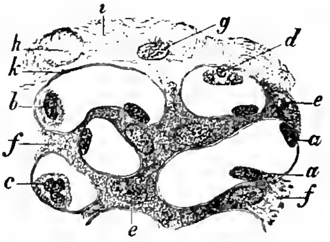

FIG. 467. SECTION THROUGH GLOMERULAR CAPILLARIES IN ACUTE NEPHRITIS FOLLOWING DIPHTHERIA.

(*The glomerulus lies near the surface of the kidney: preparation hardened in alcohol, stained with alum-carmine and eosin, and mounted in Canada balsam:* × 350)

a nucleus in capillary-wall
b swollen and loosened endothelial cell
c cell with three nuclei
d cell with nuclear fragments
e normal glomerular epithelium
f disintegrating glomerular epithelium
g nucleus of a detached epithelial cell
h vesicular (degenerate) epithelial cell
i coagulated albumen
k denuded capillary-wall

Leucocytes are often extravasated into the intracapsular space from the glomerular vessels, and mingle with the other contents. In haemorrhagic nephritis the capsule (Fig. 476 *f*) and the tubule leading from it are often tightly distended with red blood-corpuscles.

The glomerular vessels may not only contain abnormal elements, but also exhibit morbid changes in their walls. In hyperaemic conditions they are fully distended with blood. Some contain an excessive proportion of leucocytes, others may appear crammed with them (Fig. 476 *b*), and others again contain a certain number of fatty cells (Fig. 468 *C d*), whose nuclei appear broken up into from two to four pieces. These cells are probably degenerate leucocytes whose nuclei have undergone fragmentation. The vascular endothelium is more or less swollen (Fig. 467 *b*), and multinuclear cells (*c d*) appear on the capillary-walls: the latter are probably altered endothelial cells (LANGHANS, NAUWERCK). According to these authorities the capillary-endothelium becomes proliferous, and the resulting multiplication of nuclei and of cells may obstruct the channels of the glomerular vessels. Sometimes, as in scarlet fever, hyaline thrombi are formed in the capillaries (VON KAHLDEN),

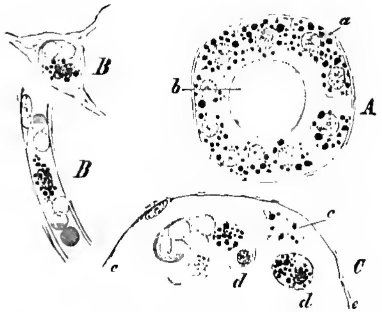

FIG. 468. FATTY DEGENERATION OF THE KIDNEYS IN DIPHTHERIA.

(*Preparation hardened in Flemming's acid solution, stained with safranin, and mounted in Canada balsam:* × 300)

A transverse section of renal tubule with fatty epithelium (*a*) and hyaline casts (*b*)
B intertubular capillary
C margin of a glomerulus with fatty epithelium (*c*) and fatty cells in the capillaries (*d*)
e capsule of Bowman

or the capillary-wall itself becomes swollen (Beer, Nauwerck, Langhans, Friedländer) and so tends to narrow its lumen. According to Langhans, hyaline deposits are occasionally formed between the proliferous endothelium and the basement-membrane of the vessel-walls, and give rise to obstruction or complete occlusion of the capillaries.

The epithelium of the renal tubules often undergoes partial necrosis, particularly in certain forms of poisoning (corrosive sublimate, chromic acid, cantharides), and in infective toxaemias. The onset of cellular necrosis can often be recognised on histological examination, even when the cells are still entire, by the fact that the nuclei do not stain or have already disappeared (Fig. 469 *c*). The secreting epithelium of the convoluted tubules is that most frequently affected, but necrotic changes may also make themselves manifest in the epithelium of the straight (efferent) tubules or of the collecting tubules. The necrotic epithelial cells are sometimes cloudy and granular, sometimes homogeneous and amorphous: at a later stage they become liquefied and dissolve.

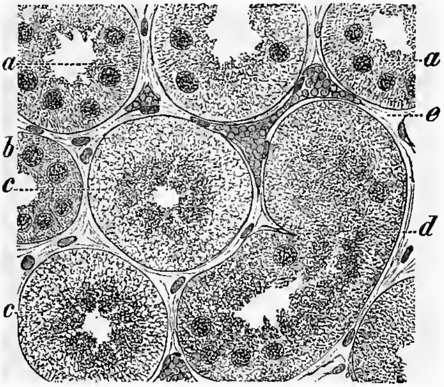

Fig. 469. Necrosis of the tubular epithelium in icterus gravis.

(*Preparation hardened in Müller's fluid, stained with gentian-violet, and mounted in Canada balsam:* × 300)

a normal convoluted tubule
b ascending limb of Henle's loop
c convoluted tubules with necrotic epithelium
d convoluted tubules with epithelium partly healthy and partly necrotic
e unaltered stroma with blood-vessels

The epithelium of the tubules passes, however, more frequently into a state of cloudy swelling (Fig. 470 *b c d*), fatty degeneration (Fig. 468 *A a* and Fig. 471 *b*), and dropsical degeneration. The changes generally affect the wide descending convoluted tubules (Fig. 471 *b*); but fatty degeneration often occurs in the limbs of Henle's loops (c_1) and in the ascending convoluted (or intercalary) tubules.

Cloudy swelling is manifested by the disappearance of the normal structure of the cell (Fig. 470 *a*), whose protoplasm becomes swollen and granular: fatty degeneration is indicated by the presence of oil-globules in the protoplasm (Fig. 468 *A a* and Fig. 471 *b* c_1), and dropsical degeneration by the sodden and swollen appearance of the cell and the formation of vacuoles in its interior.

Desquamation (Fig. 468 *A a* and Fig. 470 *d*) and finally fragmentation and disintegration of the cells are apt to be associated with all the forms of degeneration, and the tubes may thus be more or less extensively denuded of epithelium. The special way in which the secretory epithelium is involved in these forms of renal disease has led to their being grouped together under the head of **catarrhal** or **acute parenchymatous nephritis**.

Side by side with these degenerative processes, or soon after their onset, proliferous multiplication of the epithelial cells (Fig. 472 *b*) often sets in, as if an attempt were being made to replace the lost epithelium: when the case ends in recovery the attempt may be regarded as successful.

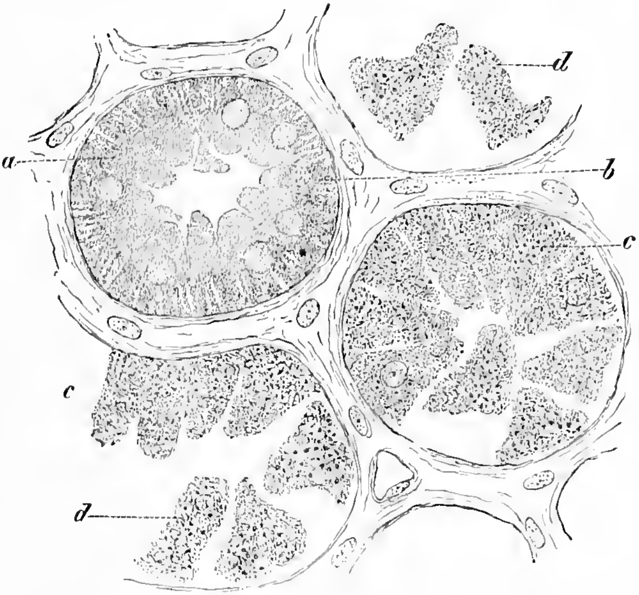

FIG. 470. CLOUDY SWELLING OF THE RENAL EPITHELIUM.
(*Preparation treated with chromic acid and ammonia*: × 800)

a normal epithelium	*c* cells in extreme degeneration
b cloudy swelling commencing	*d* loose and degenerate epithelium

The renal tubules always enclose abnormal contents at one part or another of their channels. These consist chiefly of desquamated and degenerate epithelium (sometimes agglutinated into epithelial casts by coagulated albuminous matter), leucocytes, hyaline and granular casts made up of transuded albumen and disintegrated cells (Fig. 468 *A b*), and after haemorrhage red corpuscles and their detritus in the form of pigment-granules and brown pigmented granular casts. Cases occur in which the production of casts is extraordinarily wide-spread and abundant, so that most of the loops of Henle and the convoluted tubules contain them, and the efflux of urine through them is thereby impeded.

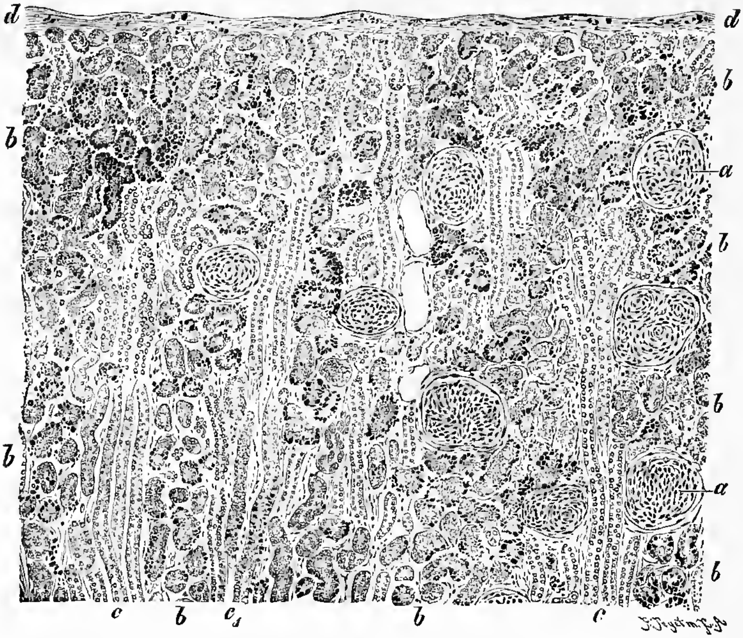

FIG. 471. ACUTE NEPHRITIS IN A CASE OF CROUPOUS PNEUMONIA.

(*See* VON KAHLDEN, *Ziegler's Beiträge* IX p. 458: *preparation fixed in Flemming's acid solution, and stained with safranin*: × 45)

a glomeruli
b convoluted tubules with fatty epithelium
c normal and c_1 fatty straight tubules
d capsule of the kidney

The intertubular connective tissue and its blood-vessels in many cases of toxaemic nephritis appear but little affected; but wherever the glomeruli and tubules are gravely and unmistakeably altered, careful histological examination always discloses morbid changes in some of the intertubular structures.

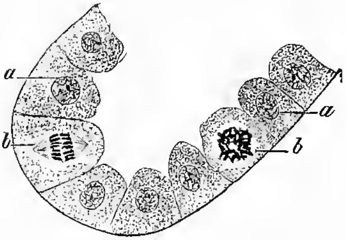

FIG. 472. PROLIFEROUS EPITHELIUM IN ACUTE NEPHRITIS.

(*Preparation placed shortly after death in alcohol, stained with haematoxylin, and mounted in Canada balsam*)

a epithelium with resting nuclei
b karyokinetic figures in proliferous cells

The interstitial alteration is in some instances limited to changes in the intertubular capillaries similar to those already described with reference to the glomerular vessels; but in other cases inflammatory exudation takes place.

When the kidney is considerably swollen and soft, oedema of the intertubular connective tissue must also be present; and the oedema is often associated with irregularly-distributed hyperaemia. In this case the tubules whose epithelium is more or less degenerate (Fig. 473 *c*), loosened, or actually exfoliated into the lumen (*d*), appear separated by broad septa (*a g*) infiltrated with liquid, fibrin, and in some places with leucocytes, which here and there are traversed by distended vessels. In later stages the interstitial hyperaemia passes away, and then multitudes of oil-globules (*a*) often make their appearance in the swollen septa. The condition might thus appropriately be described as **diffuse exudative nephritis,** or as inflammatory oedema of the kidney.

This diffuse oedema is only in rare cases so marked that its

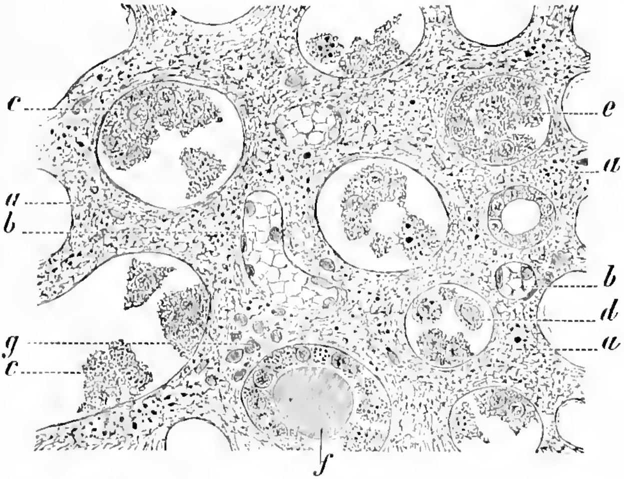

FIG. 473. DIFFUSE EXUDATIVE NEPHRITIS.

(From a man who died on the tenth day of an attack of purulent mediastinitis and pleurisy with nephritis: preparation treated with perosmic acid, and mounted in glycerine: × 350)

a infiltrated stroma, beset with fibrinous granules and filaments and a few oil-globules
b capillaries
c epithelium of the convoluted tubules, somewhat fatty and desquamating
d detached epithelium in Henle's loop
e granular and fatty detritus in Henle's loop, whose epithelium is cloudy but not yet detached
f hyaline cast in a convoluted tubule
g leucocytes

indications are readily demonstrable under the microscope, and then generally in cases of nephritis due to septic infection. More commonly the changes in the vascular stroma take the form of circumscribed cellular infiltrations of the interlobular and circumglomerular tissue, a condition which we may call **acute disseminated interstitial nephritis.**

The cellular infiltration (Fig. 474 *m*) makes its appearance chiefly in the neighbourhood of the stellate (*g m*) and the interlobular veins (*m*), and is usually so intense in these parts that in stained preparations the infiltrated patches are easily discernible even under a low power of the microscope.

The cellular aggregations are most numerous in the outer zone of the cortex, and in the boundary zone between the cortex and medulla, the middle parts of the cortex being seldom much affected. The glomeruli that lie within the region of inflammatory infiltration are generally surrounded by infiltrated cells, the latter often accumulating in a dense mass round their capsules.

The tubular epithelium is frequently altogether normal, even in the centre of the inflamed region, or it is at most a little

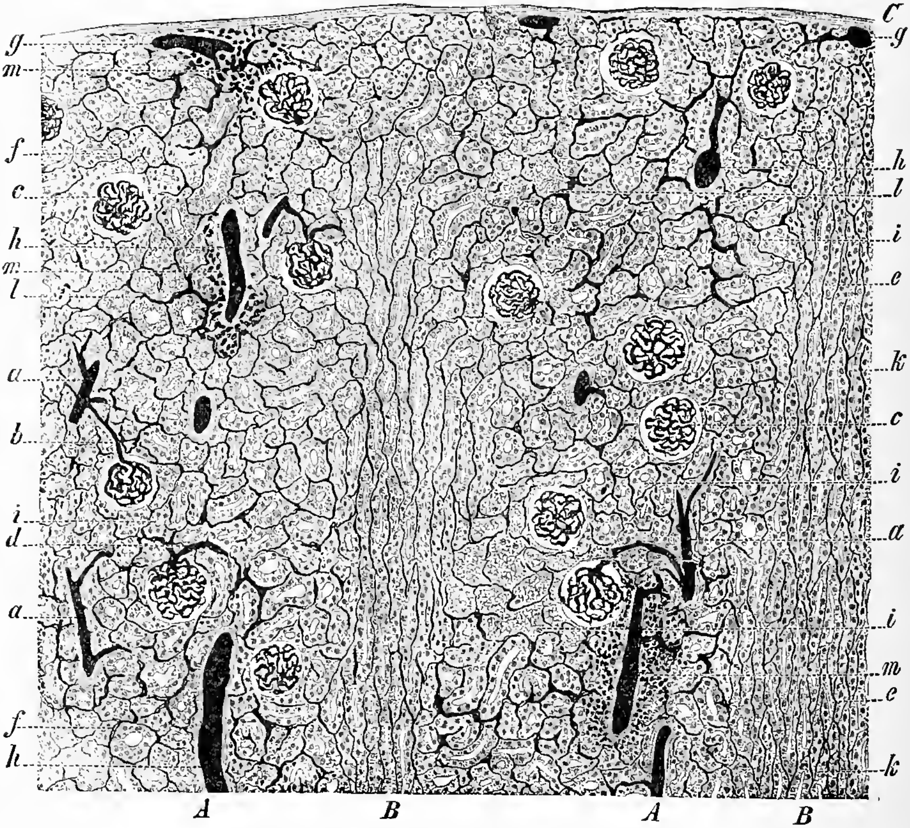

Fig. 474. Recent acute interstitial nephritis.

(Section through the outer half of the cortex: arteries injected with gelatine and Prussian blue: preparation stained with alum-carmine, and mounted in Canada balsam: × 32)

A labyrinth *B* medullary rays *C* capsule

a interlobular artery
b afferent arteriole
c glomerulus
d efferent arteriole
e capillaries of the medullary rays
f capillaries of the labyrinth
g stellate veins
h interlobular veins
i convoluted tubules
k straight tubules (Henle's loops and collecting tubes)
l degenerate convoluted tubules
m cellular infiltration round the interlobular veins

cloudy, but the normal form of the cells is retained and their nuclei continue to stain well. In other cases the epithelium undergoes unmistakeable cloudy swelling with a tendency to necrosis, especially in the convoluted tubules (Fig. 474 *l*).

The tubules within the region of cellular infiltration sometimes contain round-cells that have migrated through the membrana propria: some of these lie free in the lumen, others are enclosed within certain of the epithelial cells.

When a renal inflammation of this type runs a subacute course, and so persists for a number of weeks, the morbid changes above described tend to become more evident and pass by imperceptible gradations into those characteristic of parenchymatous nephritis (Art. 292).

References on the Morbid Anatomy of Acute Nephritis (see also Arts. 288 and 289).

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291. **Acute disseminated suppurative nephritis**, of haematogenous origin, is a condition resulting from the invasion of the kidney by certain bacteria, in particular by the pyogenic micrococci: it is in general of a metastatic nature, and consecutive to septic endocarditis or other septic affection elsewhere in the body.

The local suppurative lesions are usually rounded in shape and are seated in the cortex; but purulent streaks are also met with in the papillae, due either to infection of the medullary vessels or to invasion of the renal tubules by micrococci from the cortex.

When the bacteria settle within the capillary loops of the glomeruli (Fig. 475 *a*) they first block up the capillary channels, then induce necrosis of the glomerular epithelium (*b*), and finally necrosis of the glomerular vessels themselves. An inflammatory reaction is thereupon set up around the glomerulus, the first effect of which is the infiltration of the surrounding connective tissue with leucocytes (*d*). There is also usually a certain amount of exudation from the intertubular venules (*f*).

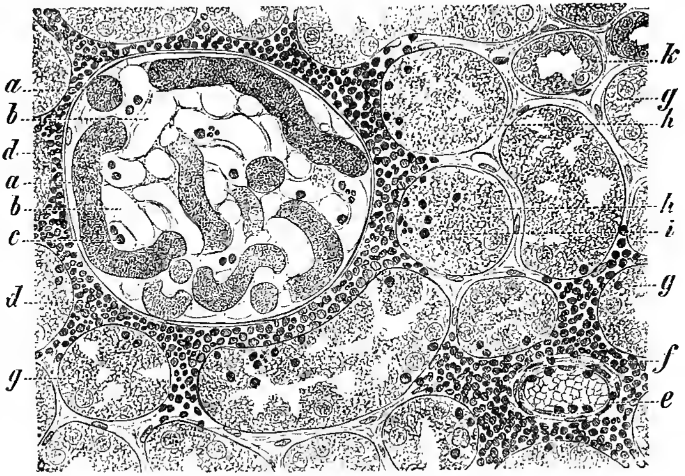

FIG. 475. DISSEMINATED SUPPURATIVE NEPHRITIS.

(Preparation stained with gentian-violet, and mounted in Canada balsam: × 200)

a	capillary loop filled with micrococci
b	empty denucleated capillary
c	leucocytes in the capillaries
d	cellular infiltration around the capsule
e	intertubular venule
f	cellular infiltration around the venule
g	convoluted tubules with epithelium partly cloudy, partly denucleated and degenerate
h	convoluted tubule with granular detritus
i	leucocytes within the tubules
k	limb of Henle's loop

The epithelium within the affected region as a rule degenerates rapidly (*g h*); part breaks down into granular detritus, part becomes necrotic and denucleated, and then desquamates. At the same time the extravasated leucocytes penetrate the tubules (*i*), and in a short time the entire region is thickly infiltrated with them. By and by not only the epithelium but also the connective tissue breaks down, and the purulent infiltration becomes an **abscess.** The size of the abscess depends of course on the extent of the initial infiltration. Small abscesses, when their pus is re-absorbed, may heal up by cicatrisation.

This form of inflammation may result in the breaking down of a large part or even the whole of the kidney so that at length nothing remains but a sac filled with pus. The latter is however not a common result of the affection now considered: it occurs much more frequently as a sequel of pyelonephritis (Art. 299, with references on suppurative nephritis).

Wide-spread suppuration of the renal tissue gives rise to catarrhal and diphtheritic inflammations of the pelvis of the kidney.

Suppurative nephritis (not due to pyelonephritis) occurs most frequently in connexion with ulcerative endocarditis and with traumatic pyaemia. It may however be associated with a great variety of diseases, such for instance as dysentery, scarlatina, typhoid fever, ulcerative pulmonary phthisis, articular rheumatism, and actinomycosis (Israel: *V. A.* 71 **1878**). The abscesses are usually punctiform or miliary; large abscesses are rare. Suppurative nephritis is not infrequently combined with embolic obstruction of the renal arteries, leading to the formation of infarcts. According to von Recklinghausen (*V. A.* 100 **1885**) infective matters occasionally reach the kidney from the vena cava by a kind of reflux peristaltic action.

292. The diseases of the kidney comprehended under the term **chronic parenchymatous nephritis** usually commence insidiously or subacutely, and lead to anasarca, which is often the first symptom that attracts the patient's notice. The urine is highly albuminous, somewhat diminished in quantity, of a turbid yellow tint, of increased specific gravity, and usually free from blood, though there are haemorrhagic varieties of the affection: in the sediment are numerous tube-casts of various sizes, white blood-cells, fatty epithelial cells, granular and fatty detritus, and fat-granule cells; red blood-corpuscles are usually few or absent, being abundant only in the haemorrhagic forms.

Recovery is rare. As a rule after the disease has lasted for months or years death ensues from increasing dropsy, cerebral oedema, pleurisy, peritonitis, uraemia, or other cause. Sometimes however the aspect of the case changes: cardiac hypertrophy and rise of the arterial pressure in the aorta cause the flow of urine to increase, its specific gravity and proportion of albumen diminish, the dropsy disappears, and the case presents the features of renal cirrhosis (Art. 294).

The aetiology of chronic parenchymatous nephritis is in general obscure, but it is almost certainly a disease of excretion, due to the elimination of the products of morbid metabolism, or of noxious substances introduced into the body from without. The fact that corresponding affections of the kidney not infrequently arise in the course of various disorders of general nutrition, such as tuberculous phthisis and diabetes, is in favour of this assumption.

The anatomical appearances associated with the foregoing changes in the urine are not always the same, and the variations described in connexion with acute nephritis are still more pronounced in the case of this form of chronic nephritis.

Naked-eye examination sometimes yields but little information regarding the actual changes that have taken place in the kidney, as there may be no perceptible swelling and but trifling deviations from the normal colour. This applies chiefly to the cases in which the textural changes are practically limited to the glomeruli, and in which the disease might from its most important characteristic be described as **chronic glomerulo-nephritis**.

Generally, however, the cortex is in some part grey, greyish-white, greyish-yellow, or white in tint, the discoloration sometimes extending over the entire thickness, sometimes affecting particular portions only, such as the internal or the external stratum, and giving the kidney now a characteristically-mottled appearance, now a uniform but abnormal hue. The term applied to the diseased organ in the post-mortem room corresponds to these variations in its appearance, and it is thus spoken of as a white, or mottled, or spotted kidney, as the case may be. If, as often happens, the kidney is also swollen, the terms large white kidney or large mottled kidney are employed to describe it.

The mottling is usually due to the alternation of pale greyish-red vascular regions with greyish-white or yellowish-white patches that contain little or no blood. Moreover, the whiter portions themselves look sprinkled or speckled, from the presence of small opaque white spots and streaks on a greyish-white somewhat translucent ground.

In contrast to the pale cortex the papillae look more or less reddened, and usually exhibit a distinct longitudinal striation. The cortical region often contains some distended veins, the turgid stellate veins appearing as red fine-rayed stars on the external surface of the kidney.

In some cases the cortex is studded more or less thickly with small circumscribed red or reddish-brown haemorrhagic patches, and these have led to the description of the corresponding affection as **chronic haemorrhagic nephritis**.

The kidney, especially if it is swollen, is usually soft in consistence. The subcapsular surface is more or less uniformly smooth; but on careful examination it is often possible to discover in it small and isolated cicatricial depressions. Some white kidneys show these in such numbers and are generally so shrunken that they approach in appearance the cirrhotic contracted kidney (Art. 294), and are accordingly referred to as **white contracted kidneys**.

The minute textural changes associated with chronic parenchymatous nephritis are also somewhat diverse; but all the types agree in this, that wide-spread degeneration of the secreting structures is associated with the excretion of urine containing albumen, epithelial detritus, and coagulated tube-casts. The predominant morbid change is fatty degeneration, both of the glomerular and of the tubular epithelium, and also of the endothelium of the blood-vessels; this change it is which when it is sufficiently extensive gives rise to the white colour of the kidney. The degenerative changes are soon accompanied by some degree of inflammatory exudation, inflammatory oedema, and cellular infiltration of the interstitial connective tissue. When the morbid process has persisted for a time, atrophic conditions invariably make their appearance also, some of the glomeruli with the corresponding tubules becoming wasted and ultimately obliterated. Slight induration of the circumscribed atrophic regions is occasionally perceptible in cases of long standing.

The glomeruli are liable to undergo all the changes already described in connexion with acute nephritis (Art. 290), and usually in a more intense degree. In many instances fatty degeneration of the glomerular and capsular epithelium is the prominent feature; in other cases this change is cast into the background by the swelling, proliferation, and desquamation of the epithelium (Fig. 476 *c g*); in others again both kinds of change are combined. With them are associated extravasations into the intracapsular spaces and the efferent tubules of albuminous liquid, often of leucocytes (*e*) also, and of blood (*f*) in the haemorrhagic varieties. From the albuminous exudations hyaline or fibrillar and granular coagula (*g*) may be deposited within the capsule of Bowman.

The glomerular vessels are apt to be compressed by the matters that fill the intracapsular space; while their endothelium often undergoes fatty degeneration, their walls sometimes become thickened and their lumina (*b*) obstructed by varying numbers of normal or fatty leucocytes or by hyaline thrombi. According to Beer, Langhans, and Nauwerck, the capillaries are liable also to be blocked by swollen and desquamated endothelium and its detritus. It is not improbable that the proliferous endothelium sometimes developes into connective tissue, as in the case of the liver under similar conditions, and thus converts the glomerular capillaries into solid impervious cords.

As the morbid change proceeds in this manner the glomeruli become obliterated; the capillary loops become impermeable (Fig. 479 *e*) and often coalesce into continuous homogeneous and almost denucleated masses. With the obliteration of the vessels the glomerular epithelium also perishes, and the entire glomerulus is at length converted into a structureless nodule, almost or altogether devoid of nuclei, and surrounded by a shrunken and somewhat thickened capsule.

In all cases the epithelium of a number of the renal tubules undergoes extreme fatty degeneration, disintegration, and desquamation (Fig. 476 *n o*). It is in the white kidney that these changes are most pronounced and wide-spread; in the greyish-red

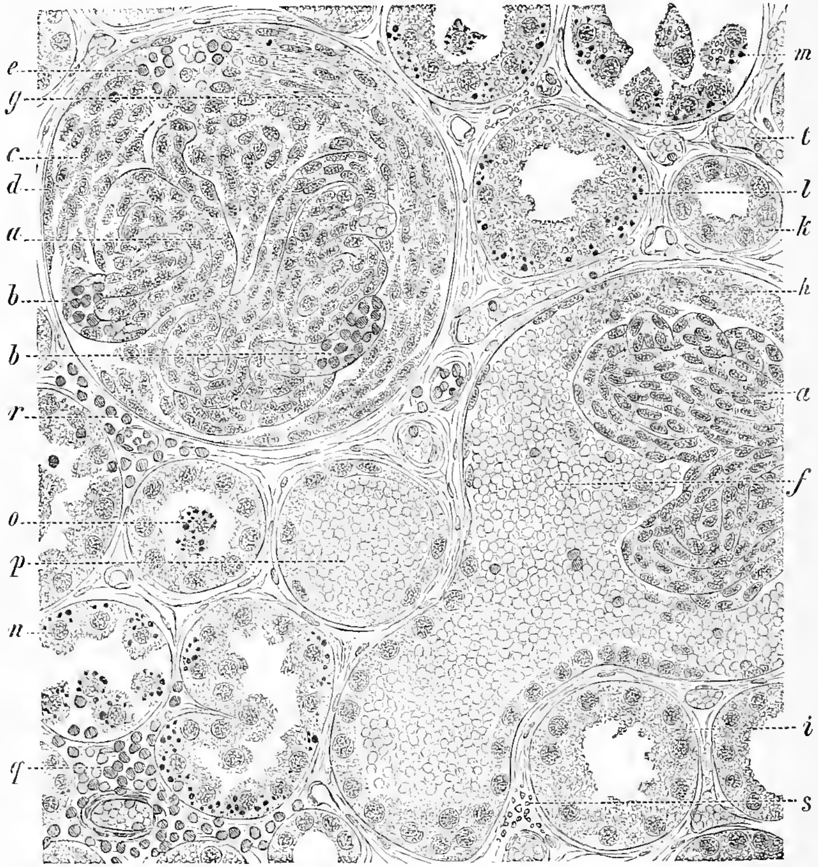

FIG. 476. CHRONIC HAEMORRHAGIC PARENCHYMATOUS NEPHRITIS.

(*Preparation hardened in Müller's fluid, stained with alum-carmine, and mounted in Canada balsam: the fatty change represented is taken from another preparation treated with perosmic acid:* × 300)

a	normal capillary loop
b	capillary filled with white blood-cells
c	desquamated glomerular epithelium
d	capsular epithelium
e	exudation consisting of red and white blood-cells and granular matter
f	haemorrhage into a capsule and its tubule
g	granular stratified exudation, containing nuclei of desquamated glomerular epithelium
h	disintegrated blood intermixed with desquamated glomerular epithelium
i	convoluted tubule
k	limb of Henle's loop
l	tubule with pigmented and fatty epithelium
m	pigmented and desquamated epithelium
n	fatty cells, some of them desquamated
o	loose fatty epithelial cells in the lumen of a normal tubule
p	tubule filled with blood
q r	cellular infiltration around venules and capillaries
s	pigment-granules in the stroma
t	capillaries filled with blood

mottled kidney they are less prominent; and in the kidney of glomerulo-nephritis they are scarcely discernible.

The fatty degeneration affects mainly the convoluted tubules, and it may also extend to the loops of Henle; but it never involves the whole of the tubules, occurring rather in scattered patches here and there, and so giving rise to the finely-mottled appearance of the cortex. The lumen of the tubules at many points contains fatty epithelial cells and their detritus (Fig. 476 *o* and Fig. 477 *f*), hyaline and granular coagula (Fig. 477 *g*), and leucocytes; and in haemorrhagic cases blood and pigment-granules (Fig. 476 *p*). In the latter class of cases some of the loosened epithelial cells not infrequently contain granular pigment (*m*).

The intertubular connective tissue is here and there the seat of more or less abundant cellular infiltration (Fig. 476 *q r*), distributed much in the same way as in acute nephritis. A certain amount of inflammatory oedema is sometimes present also.

Both the infiltrated and the non-infiltrated parts of the stroma are often beset with numerous oil-globules, some free and others enclosed in round-cells; and fatty cells can generally be seen within the intertubular capillaries. In haemorrhagic nephritis the stroma is also studded with pigment-granules (*s*).

In many cases the intertubular strands of the stroma are here and there broadened into tracts of proliferous connective tissue abundantly infiltrated with round-cells; sometimes indeed the stroma undergoes active and extensive hyperplasia, and the capsules of the obliterated glomeruli are more or less thickened. This capsular hyperplasia is most evident in kidneys with cicatricial depressions on their surface, showing that as the glomeruli became obliterated some degree of atrophy took place in the parts about them; and accordingly the thickened and condensed tissue encloses a number of slender atrophic tubules.

In virtue of these interstitial changes the diseased kidney approaches more and more nearly to the condition described by the term cirrhotic contracted kidney (Art. 294). Well-marked cases may indeed be quite excusably classed with the latter: for while a contracting white kidney, if treated by methods that are specially adapted to display fatty change, may fairly be regarded as the result of parenchymatous nephritis, in particular sections of it the fibrous hyperplasia and contraction of the atrophic regions may be so distinct, and the fatty changes so obscured by treatment with alcohol, oils, and Canada balsam, that the appearances are essentially those of an interstitial affection, and give grounds for classifying the case as one of interstitial nephritis issuing in cirrhosis and contraction.

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293. **Amyloid degeneration** of the kidney is a disease closely related to parenchymatous nephritis; it is therefore accompanied by degenerative changes in the epithelium of the glomeruli and tubules, and in many cases by inflammatory infiltration of the stroma, or by disseminated atrophy and obliteration of the secreting structures. The distinction from parenchymatous nephritis consists in the amyloid change that supervenes in and about the vascular structures, and gives the morbid condition its peculiar features.

When the epithelial degeneration, which is essentially of a fatty nature, is well-marked, the organ assumes the appearance of a large white kidney. Less extensive epithelial degeneration allows the tint of the blood and the semi-translucent texture of the renal parenchyma to be more apparent; but even in such a case the kidney is usually much paler than a healthy kidney, while at the same time it has a yellowish or greyish-yellow or greyish-white hue, and is abnormally soft. Sometimes on section the cortex shows streaks of red on a pale white-spotted ground. The glomeruli are visible as red or pale-tinted spherules, and look somewhat lustrous and translucent. If atrophic changes have already supervened, the surface of the kidney is pitted with small cicatricial depressions, and sometimes is even granular in appearance. The medullary region is usually streaked with red, but occasionally it is pale like the cortex.

The slighter degrees of amyloid degeneration cannot be detected by mere inspection. But if the cut surface is carefully washed and then treated with solution of iodine, of iodine and sulphuric acid, or of methyl-violet and acetic acid, dots and streaks of a brown (with iodine) or a reddish-violet colour (with methyl-violet) make their appearance in the parts of the vessels that have undergone amyloid degeneration, and chiefly about the glomerular capillaries and the afferent arterioles. More advanced degeneration gives the affected parts of the surface of section a lardaceous or bacon-like translucency, and increased firmness to the touch. In extreme cases the kidney is beset with broad tracts of firm amyloid substance and becomes almost rigid in consequence, while its cut surface is mottled with alternate semi-translucent and opaque-white patches.

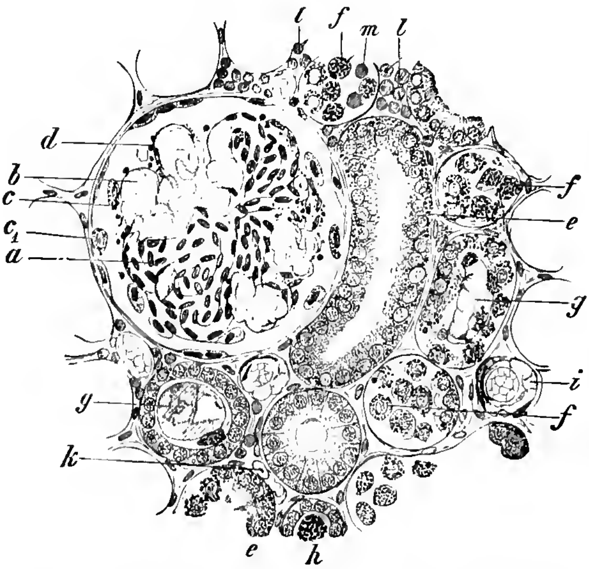

FIG. 477. AMYLOID KIDNEY WITH FATTY DEGENERATION.

(Preparation hardened in Müller's fluid, and treated with perosmic acid and methyl-violet: × 300)

a	normal capillary loop
b	amyloid capillary loop
c	fatty glomerular epithelium
c_1	fatty capsular epithelium
d	oil-globules lying on the capillaries
e	fatty epithelium *in situ*
f	loosened fatty epithelium
g	hyaline tube-casts
h	fatty tube-cast (transverse section)
i	amyloid arteriole
k	amyloid capillary
l	cellular infiltration of the connective tissue
m	round-cells within a urinary tubule

The degeneration affects mainly the vessels of the glomeruli, whose walls thereby become thickened and homogeneous (Fig. 477 *b*). At first the altered patches are scattered irregularly, but soon they coalesce, and at length the entire glomerulus is transformed into an aggregation of homogeneous blocks. Vessels that are thus entirely degenerate are of course impervious to the blood.

After the glomeruli the parts most liable to be affected are the walls of the afferent arterioles or *vasa afferentia* (*i*) and the interlobular arteries, and next the arteries of the medulla. In the end the change may extend over the greater number of the veins and capillaries of the cortex, and even involve the membrana propria of the urinary tubules. These structures are all rendered thick, translucent, and homogeneous, and yield the familiar amyloid reactions.

All the epithelial elements of the kidney, tubular (*e f*), glomerular (*c d*), and capsular (c_1), may undergo fatty degeneration. The fatty cells are in part cast off (*f*), and ultimately break down. The tubules accordingly contain various products of disintegration of the epithelium (*h*), together with hyaline casts (*g*), and not infrequently leucocytes also (*m*). The tube-casts are sometimes very soft and transparent, sometimes firm and waxy-looking, as if composed of amyloid substance. When treated with iodine these casts become browner than the unaltered tissue about them, but they do not yield the typical amyloid reactions.

In the meshes of the intertubular connective tissue we frequently find cellular infiltrations (*l*). Sometimes too there is a certain amount of local fibrous hyperplasia and induration.

As to the fatty degeneration which accompanies the amyloid change in the kidney we must assume that it is mainly the effect of the same agencies as give rise to the latter; though no doubt the disturbances of circulation occasioned by the amyloid deposits have something to do with inducing the fatty degeneration. The interstitial inflammatory changes too are probably another effect of the causes that produce the amyloid degeneration.

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STEWART, GRAINGER: *Bright's diseases* Edinburgh **1871**

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294. **Chronic interstitial nephritis** is a disease characterised by the following features—increased flow of pale slightly albu-

minous urine of low specific gravity; sediment containing few formed elements, pale hyaline tube-casts, white blood-cells, and occasionally a few red corpuscles; anasarca absent; the heart hypertrophied; the fundus of the eye affected by a special form of neuro-retinitis (Art. 369).

The disease begins either as an acute nephritis or very gradually and insidiously, the first symptoms being disorders of digestion, visual disturbances, palpitation of the heart, cardiac distress, etc. After a duration of years death ensues from such causes as cardiac failure, dropsy, cerebral haemorrhage, uraemia, purulent inflammations of the serous membranes, etc.

Its aetiology in many cases, when the process begins insidiously, remains undiscovered. In other cases it is definitely associated with disorders of metabolism (as in gout), or with chronic toxaemic conditions (as in lead-poisoning). The forms that begin acutely are generally such as follow certain specific infective diseases, and it is not improbable that some of those that begin insidiously are in reality due to the after-effects of acute infections.

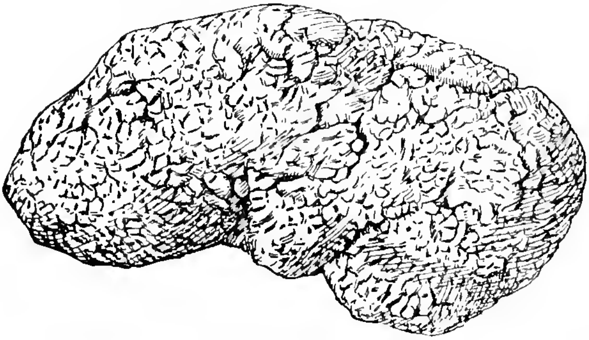

FIG. 478. CIRRHOTIC CONTRACTED KIDNEY WITH FINE GRANULATIONS.
(*Natural size*)

The post-mortem appearance of the kidney in chronic interstitial nephritis varies with the stage of the disease at which the organ is examined. If the textural changes are far advanced it is described by the term **cirrhotic contracted kidney**; its size is diminished, often very much diminished; the capsule is adherent; and the subcapsular surface invariably granulated. The 'granulations' may be generally fine or coarse, and equal or unequal in individual size (Fig. 478 and Fig. 480 *A*).

The tint of the protuberant granulations varies greatly, depending upon the amount of blood present in the cortex and on the degree of fatty degeneration in the epithelium. It is usually bright greyish-red; sometimes however it is grey, or mottled with grey and white, or almost wholly white and opaque. The intergranular depressions and contractions are usually somewhat redder.

The renal tissue is dense and tough, the cortex is more or less thinned, and the medullary papillae are often short and apparently truncated. The colour of the cortex on section corresponds with that of the surface; the medullary region is generally redder, but not infrequently it has much the same tint as the cortex.

When at the time of examination the characteristic textural changes have not yet become extensive or well-marked, the kidney is usually but little if at all reduced in size; in certain cases indeed it is somewhat swollen. Should older lesions exist in the generally less-altered cortex, they are usually indicated by more or less deep cicatricial contractions or depressions of the surface. Beneath these surface depressions the cortex is invariably thinned, while in other places it may be unaltered or even slightly increased in thickness, but the broadening is never considerable.

The microscopical appearances vary with the severity and extension of the morbid process; but in essence they always indicate partial atrophy of the secreting structures (Fig. 479 *c d h i*), hyperplasia of the interstitial connective tissue (*a k*), disseminated cellular infiltration (*c*), and usually also degenerative change in the epithelium. In other words the textural changes are the same in kind as those met with in chronic parenchymatous nephritis, but their distribution and relative intensity are different, atrophy of the secreting structures and induration of the fibrous stroma being the predominating features, and these give to this form of chronic indurative nephritis its peculiar distinctive character. It should however be noted that transitional or intermediate forms are met with, in which the epithelial structures are more gravely affected; these indicate that in some instances interstitial nephritis may differ from the chronic parenchymatous variety rather in degree than in kind.

The earliest changes in the connective tissue consist in the appearance of patches of cellular infiltration (*l*), composed of proliferous connective-tissue cells and leucocytes. Then the intertubular connective tissue (*k*) becomes more or less notably increased and appears distinctly fibrous.

The capsules of the glomeruli in the affected region are in general considerably thickened, and appear to be made up of nucleated fibrous tissue arranged in concentric layers (*a*). It is however to be noted that the amount of thickening varies greatly: in some cases it is enormous, in others very slight.

The tunica adventitia of the blood-vessels enclosed in the hyperplastic tissue (*n o*) is usually more or less thickened. Sometimes the thickening extends to the inner coats also, in particular to the intima, and leads to obstruction of the vessel. A certain number of the intertubular capillaries always become impervious as the morbid change proceeds.

The glomerular epithelium in recent cases is seen to be swollen, or loosened from the basement-membrane and desquamated (*f*), though this change is seldom so marked as in the forms of parenchymatous nephritis already described: it is also rare for the capsular epithelial cells to show much sign of multiplication or of desquamation. The glomerular capillary loops lose their epithelium (*c*) very early in the process, and are transformed into pale homogeneous finely-granular denucleated structures (*d*), which are impervious to the blood or to artificial injections.

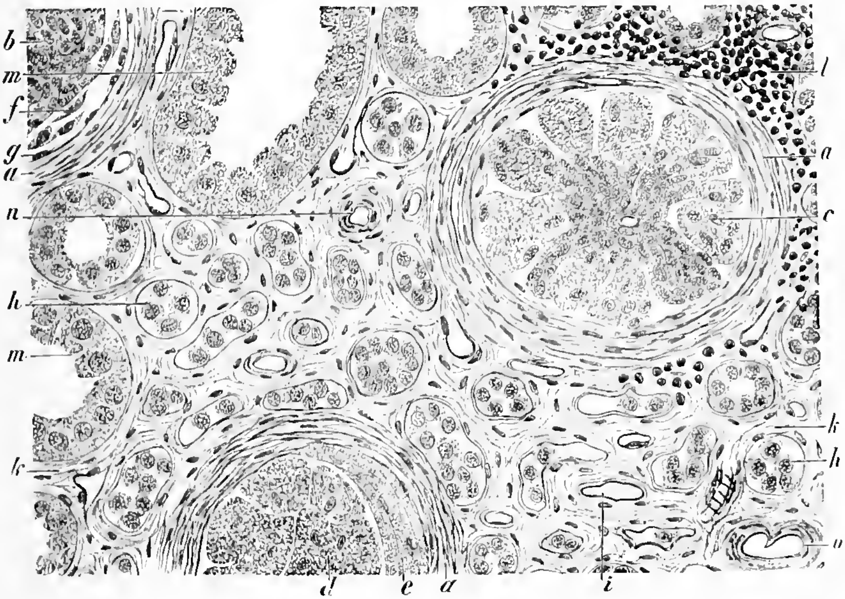

FIG. 479. INFLAMMATORY INDURATION AND ATROPHY OF THE RENAL TISSUE (CIRRHOTIC CONTRACTED KIDNEY).

(Preparation hardened in alcohol, stained with carmine, and mounted in Canada balsam: × 250)

a	capsule of Bowman thickened and fibrous
b	normal glomerulus
c	glomerulus with vessels partly obstructed and hyaline, the epithelium being nearly all destroyed
d	obliterated glomerulus
e	intracapsular coagulated mass composed of fibrinous exudation and desquamated epithelium, with interspersed nuclei
f	glomerular epithelium loosened and desquamated
g	capsular epithelium
h	collapsed tubule with atrophic epithelium
i	collapsed and denuded tubule
k	hyperplastic fibrous stroma
l	cellular infiltration
m	normal tubule somewhat dilated
n	vas afferens (arteriole)
o	small vein

During the progress of the disease the glomeruli excrete an albuminous liquid, which usually flows off into the renal tubules; in some cases however it coagulates in the presence of the desquamated epithelium within the capsule into the nucleated and stratified fibrinous masses (*e*) already described as surrounding the glomerular tuft like a hood. The albuminous liquid extravasated into the capsular space sometimes contains red and white blood-corpuscles.

The tubular epithelium undergoes the same forms of degeneration as we have described in connexion with parenchymatous nephritis, though the degeneration is usually less intense and less wide-spread.

By the time the new fibrous tissue has been formed at a particular spot the corresponding renal tubules are usually advanced in atrophy. The lumen is narrowed, and the secreting epithelium represented by small cubical cells lining the walls or lying loose within the lumen (*h*). Many tubules are entirely collapsed, their epithelium having wholly disappeared (*i*).

The contents of the unaffected tubules are the same as in parenchymatous nephritis, though fewer of them contain tube-casts and masses of epithelial detritus. Haemorrhages and pigmentary deposits are likewise less common.

If the kidney has reached the stage described as granular contracted kidney, its cortex is traversed by fibrous strands (Fig. 480 *B*), with islands of less-altered or persistent normal tissue lying between them (*A*).

The fibrous strands start from the cicatricial depressions of the surface (*B*) and run towards the bases of the medullary papillae, being interconnected by numerous transverse bands: thus in a section perpendicular to the convex surface the islands of renal tissue they enclose appear more often round or oval than elongated. The strands run as a general rule along the course of the veins, though they frequently ramify without any apparent regularity through the labyrinth. The more numerous they are the smaller of course are the islands enclosed in their meshes, and the smaller also are the granulations that project from the surface.

The fibrous strands traversing the cortex always enclose atrophied and collapsed tubules (*e f*) and obliterated glomeruli whose capsules are more or less thickened (*h i*). These strands are thus simply portions of renal tissue of which the secreting structures have been rendered functionless and the stroma hyperplastic by chronic inflammation. Here and there a tubule or a glomerulus may persist within the indurated region, while some of the tubules are dilated into little cysts by the retention of already-secreted urine (*c*). Cases not infrequently occur in which this kind of dilatation affects a considerable number of the tubules, with the result that the kidney is closely studded with cysts varying in size from that of a millet-seed to that of a cherry (cystic granular kidney), and is thereby increased in size. The condition is probably due to the fact that the intertubular tissue becomes diseased at a time when the glomeruli are still intact.

The islands of persistent secreting tissue may present a normal appearance (*a*). More frequently however certain of the tubules and glomeruli show signs of compensatory hypertrophy (*b*). Some of the epithelial cells are invariably fatty, though the extent of this change varies much in different cases. Here and there too we find patches of cellular infiltration (*k*), a sign that the inflammatory process is kept up.

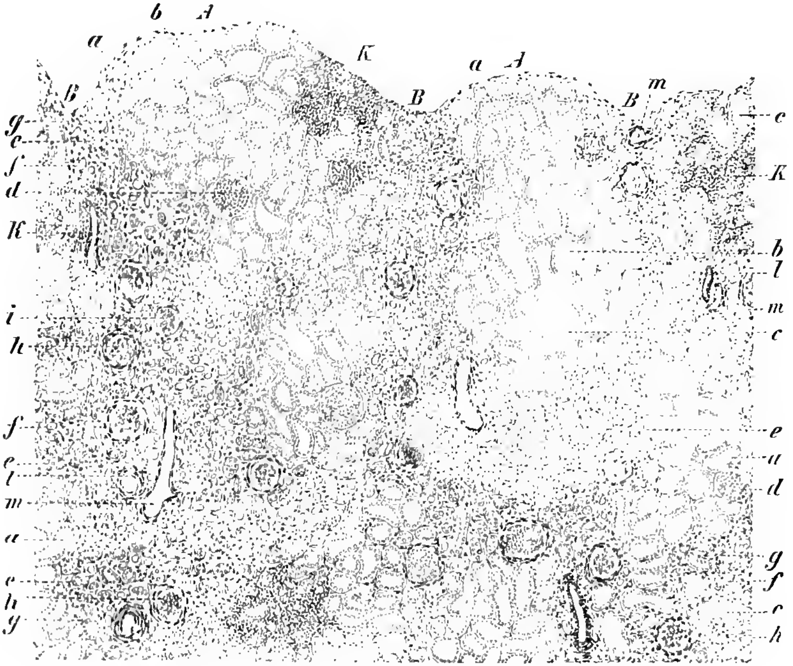

FIG. 480. CIRRHOTIC CONTRACTED (OR 'GRANULAR') KIDNEY.

(*Vertical section through the outer zone of the cortex, stained with alum-carmine, and mounted in Canada balsam:* × 40)

A	persistent remnants of renal tissue giving rise to 'granulations' on the surface
B	cicatricial bands giving rise to depressions and contractions
a	normal tubules
b	dilated tubules
c	cysts
d	normal glomeruli
e	atrophied and collapsed tubules filled with loose epithelium
f	atrophied empty tubules
g	hyperplastic fibrous tissue
h	atrophied glomeruli with thickened capsules
i	the like with normal capsules
k	cellular infiltration
l	arteriole
m	venule

Both in the cortex and in the medulla are seen tubules containing hyaline casts, or masses of shed epithelium and extravasated leucocytes.

The induration of the intertubular stroma and the loss of the glomeruli involve the obliteration of a considerable portion of the vascular system of the cortex. The vessels passing into the medullary zone consequently become dilated, though the channels thus opened up never fully compensate for the loss of the cortical vessels.

The cirrhotic and the arteriosclerotic contracted kidneys (Art. 282) represent closely-related conditions, and it is impossible to draw any sharp distinction between the two forms. This arises from the fact that atrophy of the glomeruli and of part of the arterial vascular system is a feature common to both. The only point of difference is — that in the arteriosclerotic contracted kidney the process is practically limited to the walls of the arterioles and glomerular vessels, and is very slow in its progress, while in indurative nephritis the fibrous hyperplasia takes place outside the vessel-walls in the connective tissue interstitial to the glomeruli and tubules, with the result that the stroma becomes indurated and contracted, and the secreting epithelium undergoes more extensive degeneration.

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CHAPTER XCVI

TUBERCULOSIS AND SYPHILIS OF THE KIDNEY

295. **Tuberculosis** of the kidney is in most cases due to haematogenous infection. In somewhat rare instances tuberculous disease of some part of the urinary tract extends upwards and invades the kidney.

Acute miliary tuberculosis and chronic local tuberculosis are the two forms of the affection.

Miliary tuberculosis is merely a local manifestation of an eruption of tubercles in the various organs of the body. Wherever the tubercle-bacilli lodge appears first a small semi-translucent greyish speck, which presently grows into a grey nodule: this then becomes white, and is often surrounded by a haemorrhagic areola. The greyish-white tint is due partly to cellular infiltration of the connective tissue, partly to cloudy swelling and necrosis of the epithelium. Within the tuberculous nodule the several tissue-elements break down and perish.

The number of tubercles appearing in the kidney is sometimes very great, sometimes small. Occasionally the eruption is confined to the region supplied by a single twig of the renal artery.

Chronic local tuberculosis of the kidney begins, like the miliary form, at the spot whither the bacilli have been carried and deposited by the afferent blood-stream. This may be either within the parenchyma or in the mucous membrane of the calices or pelvis, and it too is often limited to the territory of a single vessel.

At this spot greyish nodules are formed, and presently become caseous. In the course of weeks or months they grow into large irregular nodes by progressive radial infiltration, while new nodules develope around them from local infection of the tissue. In the mucous membrane of the renal pelvis the process extends partly as a diffuse infiltration, partly as a nodular eruption. The nodes in the kidney and the infiltrated tissue in the mucous membrane sooner or later become necrotic, and then disintegrate.

After a time the tissue of the kidney is studded with a varying number of grey nodules and yellowish-white opaque nodes, the larger of which are softened and excavated (Fig. 481 *a*). The medullary papillae are many of them wholly or partially caseous or already broken down, and the pelvis is in many places continuous with the tuberculous excavations of the parenchyma. The surface of the infiltrated and thickened mucous membrane of the pelvis is beset with necrotic yellowish sloughs and ulcers, or the entire wall of the pelvis is uniformly infiltrated and thickened (*b*), and its innermost layer converted into a cheesy necrotic ulcerous mass.

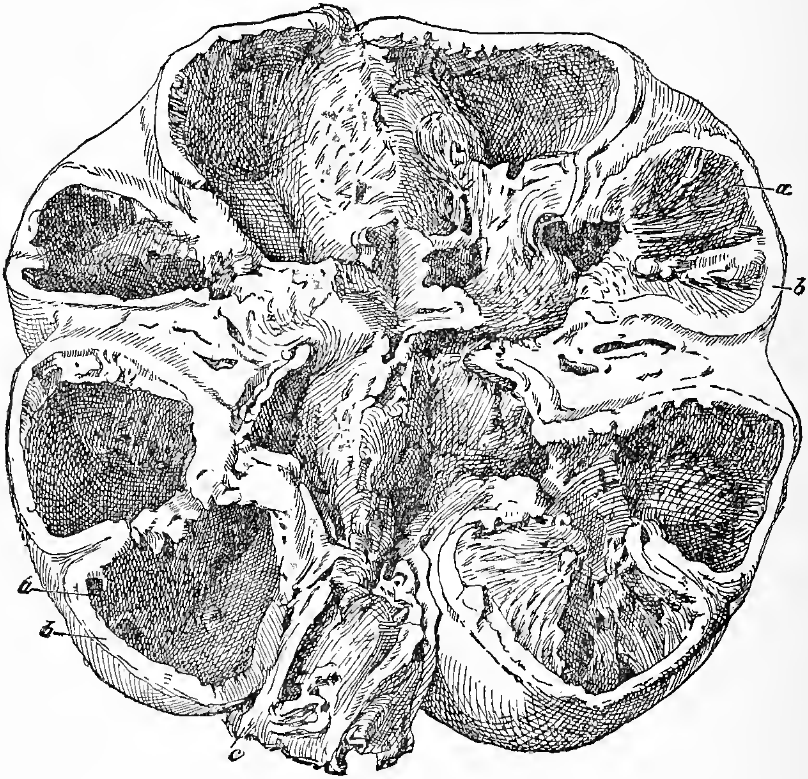

FIG. 481. ADVANCED TUBERCULOSIS OF THE KIDNEY.

(*The kidney is laid open by a longitudinal incision: two-thirds of the natural size*)

a tuberculous excavations
b tuberculous granulation-tissue
c ureter with infiltrated and ulcerated mucosa and submucosa

The tuberculous process frequently extends also to the ureter (*c*), transforming it into a more or less gristly tube with thickened walls. The inner surface is either throughout white, necrotic, and ulcerous, or it is grey and infiltrated, with scattered patches of necrosis and ulceration.

In the more advanced stages the kidney usually appears enlarged, the capsule adherent, and the surface frequently rough and tuberous. Caseous and granular detritus occupies the pelvis.

Rokitansky, Beselin, and others have observed among the detritus small shreds with a nacreous lustre, composed of squamous-looking epithelial cells, cholesterin, and inspissated pus, and not unlike cholesteatoma in appearance. The pelvis is enlarged by the excavation of the renal tissue, and to some extent by retention of urine. In extreme cases the entire kidney is destroyed, nothing remaining but a thick-walled sac.

As a rule both kidneys are affected, though it is common to find the process much more advanced in one kidney than in the other.

Syphilitic affections of the kidney exhibiting any special characteristic features are not common. Renal inflammation referable to the influence of the syphilitic virus is however occasionally observed, and is characterised by the formation of coarse cicatricial fibrous tissue and of caseating gummata resembling those that occur in the liver.

In congenital syphilis induration and contraction of the kidney have been recorded in certain rare cases. Moreover, the development of the secretory structures of the kidney may be retarded by intrauterine syphilis (Ströbe), and its fibrous tissue may become abnormally abundant.

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CHAPTER XCVII

CYSTS, TUMOURS, AND ANIMAL PARASITES OF THE KIDNEY

296. Kidneys otherwise normal occasionally contain isolated smooth-walled **cysts** varying in size from that of a pea to that of an apple. Such cysts are due to the retention of secretion in renal tubules whose glomeruli are still functionally active but whose channel is at some point occluded. They accordingly contain a clear and colourless or slightly-yellowish liquid.

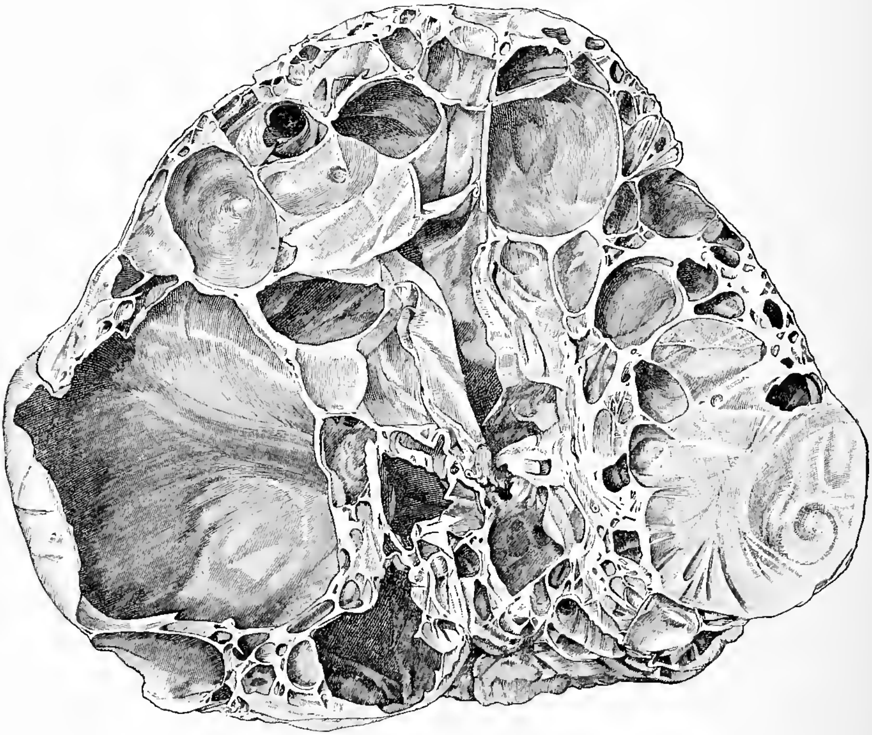

FIG. 482. CYSTOMA OF THE KIDNEY.

(*Transverse section: eleven-fourteenths of the natural size*)

Multiple cysts varying in size from that of a pin-head to that of a pea are often found in kidneys that are morbidly altered by chronic nephritis or arteriosclerotic contraction. Their contents are either liquid or colloid, according as they are the result of persistent secretion in the tubules concerned or of morbid processes in the epithelium (Fig. 461). Sometimes contracted kidneys are thickly beset with small colloid cysts of this kind.

Cystic degeneration of the kidney, leading to the formation of large renal tumours, usually results in the transformation of the greater part of the organ into a bulky tumour made up of an aggregation of cysts varying from the size of a pea to that of an apple (Fig. 482). As a rule little or none of the normal glandular parenchyma persists between the cysts, and the portions that remain are gravely altered in structure and disposition.

This cystic transformation is met with both in adults of advanced years and in new-born infants and children: in the latter case the condition is described as **congenital cystic disease.** In some uncommon cases the infantile cystic kidney is more or less markedly enlarged, and so closely honeycombed with minute cysts that it looks on section like a fine-meshed sponge (Fig. 483).

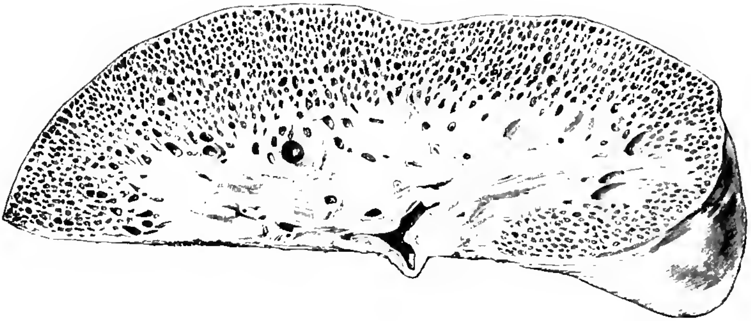

FIG. 483. CONGENITAL CYSTIC KIDNEY.

(*The kidney is* 12·5 *centimetres long,* 9 *centimetres broad, and* 5 *centimetres thick, and its cortex is spongy in structure: natural size*)

According to NAUWERCK, HUFSCHMID, VON KAHLDEN, and others, the cystic kidney must in some cases be regarded as a neoplastic formation or **adenocystoma**, inasmuch as the cysts are not derived from renal canals, but from atypical gland-like tubules, which may make their appearance either in normal or in malformed kidneys. In the congenital forms the cysts (Fig. 484 *e*) may be derived not only from tubules (*b*) but from the glomerular capsules also (*c*), and their walls sometimes enclose glomerular tufts of vessels (*d*), which by secreting liquid contribute to the dilatation of the cyst. In these cases there is manifestly some textural malformation of the kidney whereby, for no very obvious reason, the renal tubules have at some point been rendered impervious. Probably this is due in certain cases to developmental anomaly, in others to intra-uterine morbid change (such as inflammation) in a kidney otherwise normally fashioned.

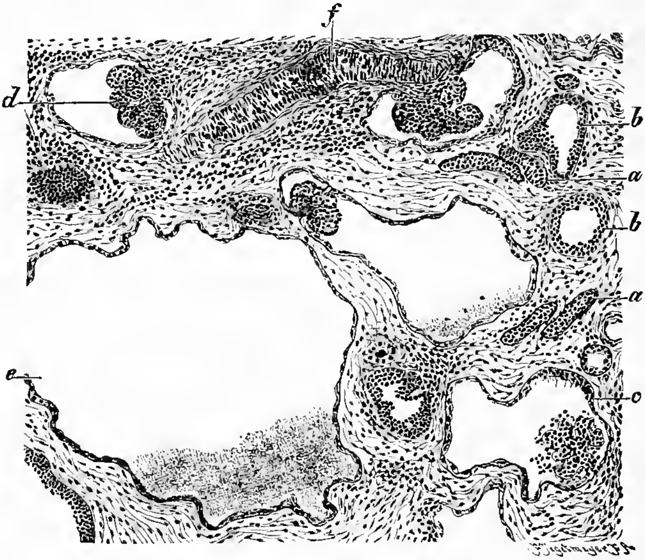

FIG. 484. SECTION FROM THE CYSTIC KIDNEY OF FIG. 483.

(*Preparation hardened in Flemming's acid solution, and stained with safranin* : × 45)

a	glaudular tubules	*e*	large cysts
b	dilated tubules	*f*	artery
c	cysts with glomeruli *d*		

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297. The **tumours** met with in the kidney include representatives of the epithelial, histioid, and teratoid types. Of the epithelial neoplasms both adenomata and carcinomata are found.

Adenoma takes the form of well-defined soft white nodes varying in size from that of a millet-seed to that of a walnut; some of them are alveolar, and others are tubular in structure. Papillary growths are occasionally developed in the gland-like tubules or acini and in the neoplastic alveoli, giving rise to the form known as papillary adenoma (Fig. 485). When liquid gathers to any considerable amount in the tubules and dilates them into cysts, the tumour is described as cystadenoma or cystoma (Art. 296, Fig. 482).

Carcinoma of the kidney gives rise to soft or hard tumours, which increase the bulk of the organ, sometimes to an enormous degree. Smaller growths involve only a portion of the kidney, and are often fairly well marked off from the surrounding parenchymatous tissue. Large tumours sometimes infiltrate and destroy the whole of the parenchyma, and the cancerous growth in certain cases extends into the pelvis of the kidney. Such growths frequently contain in their substance softened and haemorrhagic patches, whence blood and cancerous detritus pass into the urine. Renal carcinoma occurs at all ages, but is relatively frequent in children. It is probable that it may develope from a pre-existing adenoma; and it occasionally arises in kidneys that have already undergone some morbid change.

Sarcoma of the kidney makes its appearance in young children or even in new-born infants as well as in older persons, and gives rise to tumours of considerable size (4 to 6 kilogrammes). They are generally soft and more or less vascular, and may belong to any or all of the cellular types characteristic of sarcoma. In many cases renal sarcoma has been shown to contain spindle-cells with something of the structure of striated muscle (rhabdomyoma), a fact which suggests that developmental anomalies of structure may have something to do with the genesis of sarcoma: the occasional occurrence of bone and of cartilage in these myosarcomatous growths is probably to be interpreted in the same sense as evidence of their teratoid nature. Alveolar angiosarcoma (endothelioma) is a special form of the neoplasm, of which in a number of cases examples have been met with in the kidney.

Fibromata are frequently met with in the kidney, and take the form of nodules of the size of a pea or smaller. Large fibromatous growths are very rare, as are also nodose **angiomata, myxomata, lipomata,** and their combinations. Vascular tumours projecting into the renal pelvis often give rise to haemorrhage and haematuria.

Tumours possessing the structure of suprarenal tissue, and accordingly described as *strumae lipomatodes aberratae renis* (GRAWITZ), are not infrequently met with in the form of white fatty-looking sharply-defined subcapsular nodes, varying from the size of a pea to that of a cherry. They manifestly consist of aberrant portions of the suprarenal body, being made up of a vascular stroma enclosing nests and rows of cells thickly studded with oil-globules. The observations of ASKANAZY, LUBARSCH, and others, render it probable that large malignant tumours resembling carcinoma in structure occasionally arise from such misplaced and degenerate embryonic remnants of suprarenal tissue.

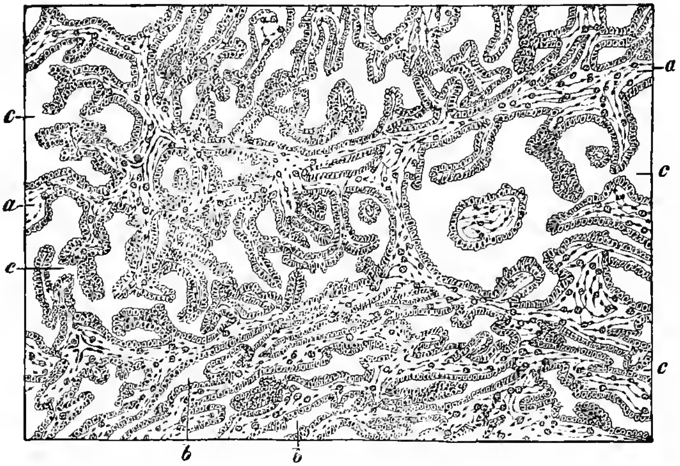

FIG. 485. PAPILLARY ADENOMA OF THE KIDNEY.

(*Preparation hardened in Müller's fluid, stained with haematoxylin, and mounted in Canada balsam:* × 30)

a stroma *b* gland-like tubules *c* dilated tubules with papillary growths

Among the **secondary growths** found in the kidney both sarcoma and carcinoma are met with; they usually form rounded nodes, that are however not always sharply circumscribed, inasmuch as the neoplastic infiltration extends by way of the intertubular vessels.

Of the **animal parasites** infesting the kidney *Echinococcus* is the most important. It forms hydatid cysts from the size of a hazel-nut to that of a child's head, with or without daughter-cysts. The cysts sometimes burst and are evacuated into the pelvis of the kidney. When the scolices die the cyst may contract, and its contents become inspissated and cretaceous.

Cysticercus cellulosae and *Pentastoma denticulatum* are very rare. When the blood contains *Filaria* a large number of the parasites reach the kidney, and may be found both outside and inside the vessels. Their presence in the kidney and in the pelvis and abdominal lymphatics occasionally gives rise to intermittent haematuria and chyluria, the urine in the latter case becoming turbid and even milky owing to its admixture with innumerable very minute oil-globules.

Eustrongylus gigas and *Bilharzia* or *Distoma haematobium* infest the pelves and ureters (Art. 299), but they sometimes induce inflammation and ulceration of the adjoining renal tissue.

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CHAPTER XCVIII

THE RENAL PELVES AND THE URETERS

298. The most important changes that take place in the renal **pelvis** and the **ureter** are those due to dilatation from retention of urine, and to inflammation. Tumours of these parts are on the whole very rare.

Dilatation of the renal pelvis and the ureter from retention of urine takes place when the outflow of urine is prevented or obstructed at any point in the urinary tract, as by impacted calculi, cicatricial stricture of ureter or urethra, enlargement of the prostate impeding the evacuation of the bladder, compression, flexure, or torsion of the ureter, valvular folds of the mucous membrane, abnormal constriction of the preputial orifice (phimosis), etc. Obstruction of one ureter causes unilateral dilatation of the corresponding pelvis and ureter: obstruction at the mouth of the bladder or in the urethra is usually followed by bilateral backward pressure and distension. Dilatation of the renal pelvis from retained urine is called **hydronephrosis**.

The extent of the dilatation depends on the completeness of the obstruction to the outflow of urine, the copiousness of the secretion, and the duration of the morbid condition. Moderate dilatation of the renal pelvis produces flattening of the papillae and stretching of the cortex of the kidney; more extreme distension compresses the cortex and flattens the tubules and glomeruli. When the pressure is long maintained it induces progressive atrophy and ultimate obliteration of the tubules and glomeruli, the connective tissue remaining intact or even becoming hyperplastic. The hydronephrotic sac thus produced may contain 10 to 20 litres of liquid, and its fibrous walls enclose but scanty remnants of secreting tissue. Even when the secretion of urine ceases, owing to the progressive atrophy of the renal parenchyma, the pelvic mucous membrane continues to contribute mucous liquid to the contents of the sac. These liquid contents are usually clear and colourless, or of a slightly-yellowish tint; but sometimes from admixture with blood and the products of its disintegration they acquire a brownish colour. In cysts of some standing cholesterin-plates and broken-down pus-cells are occasionally found. When the liquid becomes puriform, the condition is described as **pyonephrosis**.

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299. Inflammation of the renal pelvis and the ureter, called respectively **pyelitis** and **ureteritis**, results from contamination by irritant substances of the urine secreted by the kidney, or from infection proceeding upwards from the contents of the bladder, this latter taking place chiefly in cases of retention and backward pressure of the urine. In rare cases the infection of the urinary tract is haematogenous.

In the production of ascending infection *Bacillus coli communis*, the pyogenic micrococci, and *Gonococcus* are the principal agents; in infection proceeding from the kidney itself any of the infective or toxic matters that are capable of inducing renal disease may be operative. Of irritant animal parasites *Eustrongylus gigas*, *Filaria sanguinis hominis*, and the ova of *Distoma haematobium* (*Bilharzia*) are the chief. Lastly, concretions and calculi lodged in the renal pelvis and ureter not infrequently give rise to local inflammation. According to their causation, therefore, we may distinguish pyelitis and ureteritis as infective or parasitic, toxic, or calculous.

Infective and toxic pyelitis and ureteritis, in the slighter forms that appear in the course of typhoid fever, scarlatina, small-pox, pyaemia, cholera, cantharides-poisoning, etc., are catarrhal in nature, and tend to pass away on the complete elimination of the injurious substances. Severe purulent or diphtheritic inflammation is usually due to *Bacillus coli communis* and the pyogenic micrococci, or less frequently to *Proteus vulgaris*. Such inflammations are usually set up by infection from the bladder; they are sometimes associated with a pre-existing hydronephrosis, and render the contents of the dilated pelvis turbid and puriform (pyonephrosis). They not only give rise to collections of pus in the urinary tract but often extend to the kidney itself, and so produce **pyelonephritis**. In this condition the accompanying inflammatory oedema causes the kidney to become swollen, sometimes to an enormous extent, and studded throughout its medulla and cortex with yellowish-white punctiform abscesses surrounded by hyperaemic areolae. If the patient survives this stage, the process may pass on to the formation of large abscesses extending to the capsule and perinephric tissue, and of new hyperplastic fibrous tissue indurating the kidney and encapsuling the abscesses. When the suppuration predominates the abscesses may burst into the renal pelvis and be thus evacuated; while the renal parenchyma is for the most part destroyed, and nothing remains in the place of the kidney but a large pus-filled sac or **pyonephrosis**.

The inflammation caused by the eggs and embryos of *Distoma haematobium* leads to induration with the formation of papillary growths, or to ulceration of the mucous membrane, which when thus morbidly altered is apt to become encrusted with urinary concretions.

Calculous pyelitis is a result of **nephrolithiasis**, or the condition in which the pelvis contains calculi (Arts. 285, 286) that damage the mucous membrane by pressure and friction and often impede the outflow of the urine. The inflammation is in general of a catarrhal kind, but when the mucous membrane is actually wounded by the calculi haemorrhagic effusion is apt to take place. In cases of long standing the walls of the pelvis are usually thickened from fibrous hyperplasia. Secondary infection may cause the calculous inflammation to become purulent.

The inflammation sooner or later extends to the renal parenchyma, and leads to swelling and cellular infiltration, terminating in suppuration or in fibroid induration. In either case some portion of the renal tissue is destroyed. The whole of it may perish in extreme cases, leaving nothing but a fibrous sac surrounding the calculi. **Perinephritic abscesses** also are frequently produced.

When a calculus of some size becomes wedged in the ureter it may occlude its channel and stop the outflow of the urine. If in consequence of this a considerable accumulation of urine takes place in the pelvis of the kidney we may have a hydronephrosis (Art. 298) superimposed on the calculous pyelitis. The retained urine often decomposes and thus intensifies the inflammation, so that it becomes purulent: in this way pyonephrosis succeeds hydronephrosis.

An impacted stone may be gradually urged forward into the bladder by the pressure of the accumulating urine, giving rise to haemorrhage, erosion, and inflammation on its way.

Suppurating ulcers of the renal pelvis may break through externally and thus enable pus to escape into neighbouring parts, such as for instance the intestine or the bladder. More often the pus escapes into the perinephric (subperitoneal) cellular tissue, and gives rise to wide-spread suppurative or septic inflammation (perinephritic abscess). Calculous pyelitis is usually unilateral, rarely bilateral.

When the pelvis of the kidney and the ureter are from any cause inflamed, small grey projecting nodules are sometimes formed in the mucous membrane. These consist of lymphadenoid tissue, and in all probability represent swollen lymph-follicles normally existing in the submucosa; but it is possible that they are first formed in the course of the inflammatory process (CHIARI, PRZEWOSKI). In other cases small cysts appear in the inflamed mucous membrane, varying in size from that of a millet-seed to that of a hemp-seed, and lined with epithelium, their contents being limpid or viscid in different cases. Their presence has given rise to the terms cystic pyelitis and ureteritis. From the researches of VON KAHLDEN, it appears probable that as PISENTI, SUTTON, CLARKE, and others have suggested, these cysts are due to the presence of a peculiar *Sporozoon*.

In rare and chronic cases (chiefly tuberculous) the epithelial cells of the urinary tract undergo a peculiar kind of cornification, and assume the form of lustrous white scales, the inner surface of the mucous membrane thus becoming specked with structures resembling cholesteatomata.

Tuberculosis of the pelvis and ureter is usually a descending affection following the elimination of tubercle-bacilli from the kidney, and is indicated by the formation of caseous granulomatous growths on and in their walls (Art. 295, Fig. 481 *c*).

Tumours of the pelvis and ureter are on the whole rare; but carcinoma and connective-tissue growths are occasionally observed in them, sometimes in the form of villous or papillary excrescences arising from their walls. Carcinoma at times appears in association with chronic calculous pyelitis.

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CHAPTER XCIX

THE URINARY BLADDER

300. **Morbid changes in the vesical contents.** The urinary bladder is the temporary receptacle of the renal secretion. When the urine is mingled with abnormal exudations from the blood-vessels, or the products of morbid change in the kidney or its pelvis, these impurities are naturally retained for a certain time in the bladder. Morbid secretions from the wall of the bladder may also be mingled with the urine. Of the formed matters thus occurring in the urine the following are the most important.

Red blood-corpuscles or their detritus come either from the kidney, the renal pelvis, or the bladder. In the former case they have in general escaped from the glomeruli as a result of disordered circulation, or of inflammation. They are rarely derived from intertubular haemorrhage. Vascular neoplastic growths in the kidney (such as carcinoma and angioma) are also apt to give rise to haemorrhage and haematuria.

When a part of the extravasated blood coagulates in the tubules the urine contains dark and opaque granular tube-casts, which enclose blood-cells or their detritus and are known as **blood-casts.**

Haemorrhage from the pelvis of the kidney is generally due to inflammation and erosion caused by renal concretions. Bleeding from the mucous membrane of the bladder occurs as a result of acute inflammatory and ulcerative processes, and also in cases of extreme venous engorgement, in scurvy, haemorrhagic small-pox, scarlatina, etc. Wounds, vesical calculi, and new-growths of the bladder-wall, such as papilloma and carcinoma, not infrequently lead to vesical haemorrhage.

White blood-cells appear in connexion with inflammatory conditions of the kidney and its pelvis, or of the bladder; they are most abundant in purulent pyelitis and cystitis. Occasionally collections of pus in the surrounding parts break into the urinary organs and passages. In tuberculous and other inflammations associated with destruction of tissue the urine contains bacilli and necrotic detritus.

In morbid conditions of the kidney renal **epithelial cells** enter the urine from various parts of the tubular system; they are usually more or less degenerate, especially when they come from the cortical region. When they are shed in abundance they often cohere together to form **epithelial casts.**

Pelvic epithelial cells enter the urine chiefly in cases of pyelitis and pyelonephritis: epithelial cells from the bladder pass into it when vesical inflammation or papillomatous tumours of the bladder-wall are present. The epithelial cells thus shed into the urine are of very diverse forms, but certain definite types (Fig. 486 *a b c*) corresponding to the several layers of the stratified vesical epithelium are constantly met with. In cases of papilloma fragments of the villous growth (Fig. 487) are apt to be detached and escape with the urine. Large numbers of **cancer-cells** often enter the urine from ulcerous carcinomata of the kidney and bladder; they are polymorphous and deviate in many respects from the typical cells of the vesical epithelium.

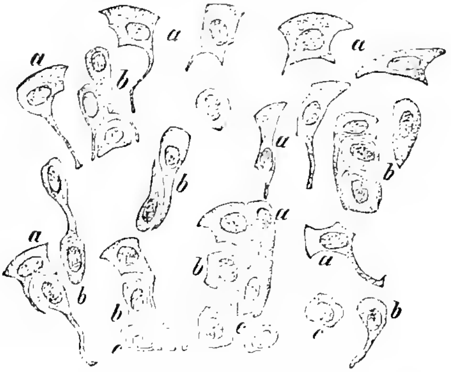

FIG. 486. VESICAL EPITHELIAL CELLS FROM A FRAGMENT OF A PAPILLOMA EVACUATED WITH THE URINE.

(× 250)

a from the superficial strata
b from the intermediate strata
c from the deepest strata

Tube-casts come from the renal tubules, in which they are formed in various affections of the kidney. They have an approximately cylindrical form, and are either hyaline and colourless, granular, or lustrous and waxy-looking with a slight yellow tinge. All forms of casts may

FIG. 487. PAPILLOMA OF THE URINARY BLADDER.
(*Teased preparation of a f*[illegible] *passed with the urine*: × 100)

have adhering to them tubular epithelial cells and their detritus in the form of albuminous and fatty granules, colourless and red blood-cells, granular urates, or crystals of uric acid and calcium oxalate.

Scolices and daughter-cysts may reach the bladder from **hydatids** seated in the kidneys or the urinary passages; and when the mucous membrane of the pelvis and ureter is beset with the ova of *Distoma haematobium* or with *Filaria*, ova and embryos of these parasites generally pass into and are discharged with the urine.

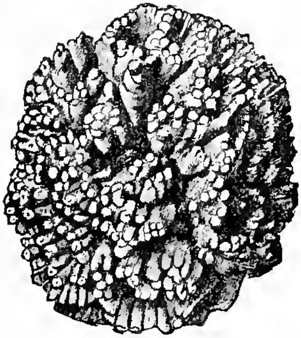

FIG. 488. CORAL-LIKE VESICAL CALCULUS COMPOSED OF CALCIUM OXALATE AND PHOSPHATE.

(*Natural size*)

Bacteria usually reach the bladder through the urethra; but they sometimes gain access to it from bacterial colonies in the kidney. Should they find in the urine a suitable medium for their growth, and escape immediate evacuation from the body, they are apt to multiply in the bladder. Some of them set up ammoniacal fermentation in the urine. **Yeast-fungi** have been known to induce alcoholic fermentation, with evolution of carbonic acid, in saccharine (diabetic) urine within the bladder.

Faeces sometimes enter the bladder when a fistulous communication exists between it and the rectum. When a dermoid cyst ruptures into the bladder, its heterogeneous contents may also mingle with the urine.

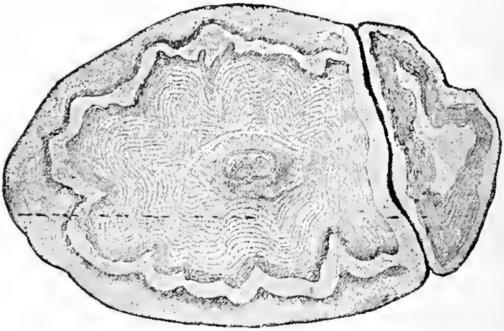

FIG. 489. SECTION THROUGH TWO CONTIGUOUS FACETTED STONES COMPOSED OF SODIUM URATE AND AMMONIUM-MAGNESIUM PHOSPHATE.

(*Natural size*)

Children and others addicted to self-abuse occasionally introduce through the urethra solid objects (lead-pencils, hair-pins, straws, and the like) into the bladder; and fragments of surgical instruments such as catheters are sometimes accidentally left within it. Bullets that have penetrated the tissues and reached the bladder have been found lying loose in its cavity.

Vesical **concretions** and **calculi** either come from the pelvis of the kidney and the ureters (Arts. 285, 286), or are formed within the bladder. In other cases foreign bodies that have entered the urinary passages from without form the nuclei of vesical calculi, which usually consist of triple-phosphate and calcium phosphate, or more rarely of uric acid and urates.

Vesical calculi are globular or ovoid (Fig. 489), and may be smooth or bossed and tuberous, or rough and even spiny (Fig. 488). When more than one are present they are occasionally facetted. Some stones are hard, others soft and friable. Often they are distinctly laminar in structure (Fig. 489) and consist of various substances in successive layers.

The presence of a stone in the bladder generally sets up inflammation, and not infrequently ulceration and haemorrhage also. Its irritation excites the bladder to frequent contraction, and often at the same time interferes with free micturition, with the result of inducing hypertrophy of the bladder-wall. At times the calculus is lodged in a diverticulum or sacculation of the wall.

Vesical calculi are classified according to their composition.

(1) Calculi consisting chiefly of uric acid or urates. Pure uric-acid stones are generally small and hard, and of a yellowish-red or brown tint. Uratic stones are seldom pure. Usually the surface layers are composed of calcium oxalate and ammonium-magnesium (triple) phosphate.

(2) Calculi consisting chiefly of phosphates and carbonates. These are composed mainly of calcium phosphate or of ammonium-magnesium phosphate, and of calcium carbonate. Stones of pure calcium carbonate are very rare. All these stones are white or greyish-white. The triple-phosphate stones are soft and friable, the others are hard.

(3) Calculi of calcium oxalate. These are hard and spiny and brown in colour.

(4) Calculi of cystin are soft, brownish-yellow, and waxy.

(5) Calculi of xanthin are of a cinnabar-red colour, with a smooth surface and earthy fracture.

301. **Inflammation** of the wall of the bladder, or **cystitis,** is in most cases caused by the presence of irritant matters in the urine; but it may be of haematogenous or traumatic origin, and is sometimes produced by the extension of inflammation from adjacent parts. Among the bacteria that cause inflammation of the bladder, the pyogenic micrococci, *Gonococcus*, *Bacillus coli*, *Bacillus tuberculosis*, *Proteus vulgaris*, and various bacilli that decompose urea, are of special importance.

Catarrhal cystitis is characterised by the occurrence of shed epithelium, pus-cells, mucus, and generally red blood-corpuscles in the urine. According to KOSSEL, the slimy character of cystitic pus is due chiefly to the presence of sodium chloride and ammonium carbonate. In recent cases the mucous membrane appears *post mortem* to be but little altered. When the secretion is purulent the membrane is covered with a film of pus, and is sometimes very much swollen. When haemorrhage has occurred the surface is of a uniform grey tint, or mottled with grey, black, and reddish-brown patches. When the mucous membrane of the bladder contains small aggregations of lymphadenoid tissue, which are not uncommon about its neck, these are apt to protrude from the injected surface as greyish-white nodules. In rare cases small cysts are formed in the mucous membrane. Purulent or putrid inflammation often extends to the submucous and the muscular layers, causing them to become infiltrated and more or less thickened. In very severe cases haemorrhagic and slaty-grey patches appear on the serous or peritoneal surface; and at length purulent or putrid exudations make their appearance in the surrounding cellular tissue (paracystitis), or on the peritoneum itself (pericystitis).

Certain irritants, such as cantharides, lead from the outset of the affection to superficial sloughing of the epithelium, which becomes detached in the form of necrotic flakes and shreds. Such infective disorders as measles, scarlatina, typhoid, septicaemia, etc., are occasionally accompanied by superficial diphtheritic exfoliation in the form of isolated yellowish patches; in other instances the exudation is croupous.

When the urine becomes ammoniacal and putrid, the epithelial layers, the connective tissue of the mucosa and submucosa, and even the muscular coat, may suppurate and become necrotic, and at length gangrenous and putrid. In this way ulceration, gangrene, and abscess of the bladder-wall are developed, and in the end the wall may be perforated, and the suppuration and gangrenous necrosis spread to the surrounding tissue.

In the severer forms of cystitis the mucous surface is frequently rough and sandy from encrusted urinary salts.

In **chronic cystitis** fibrous hyperplasia of the coats of the bladder, with true hypertrophy of its muscular fibres, is of common occurrence.

Tuberculosis of the bladder begins with the formation of grey nodules surrounded by a zone of hyperaemia; these subsequently enlarge and turn yellow, and sooner or later break down into ulcers. The ulcers have a cheesy infiltrated floor, and their borders are hyperaemic. They increase in size by progressive marginal disintegration and by coalescence, and in this way are formed large sinuous ulcerations involving a considerable part of the mucosa and submucosa. Vesical tuberculosis is usually accompanied by tuberculosis of the pelvis of the kidney, or, in the male, of the genital apparatus.

Persistent venous engorgement leads to varicose dilatation of the veins of the mucous membrane of the bladder. They are sometimes referred to as **vesical haemorrhoids**, and now and then obstruct the evacuation of the bladder or give rise to haemorrhage.

Amyloid degeneration of the vesical mucous membrane is not rare, but as a rule it is not apparent to the naked eye. In very rare instances the amyloid deposits lead to diffuse induration of the mucosa and submucosa.

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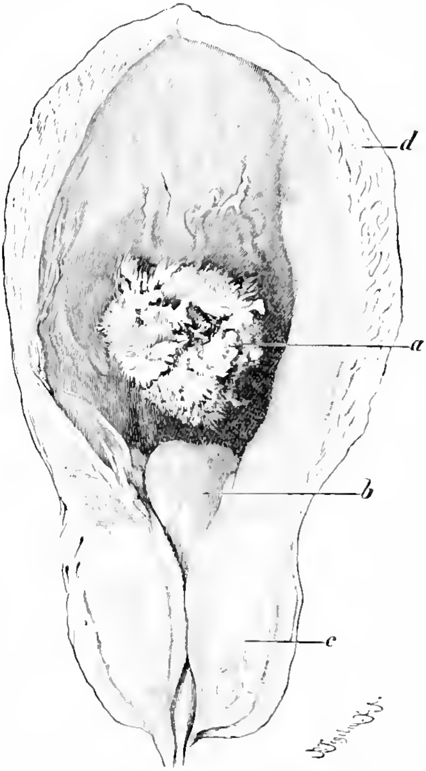

Fig. 490. Papillomatous fibroma of the bladder with hypertrophy of the prostate.

(*Five-sixths of the natural size*)

a fibroma
b c enlarged prostate
d thickened and inflamed wall of the bladder

302. The commonest of the **tumours** of the bladder is the so-called **villous cancer**, or vascular papillomatous **fibroma**. It consists of a number of long and slender villi or papillary growths (Fig. 490) springing generally from a comparatively narrow base, but sometimes

from a broad one. Each villus consists of vascular connective tissue, and is covered by a very thick layer of epithelial cells (Fig. 490). The growth does not extend into the deeper layers of the mucous membrane, but grows out of it, and sometimes attains the size of a small apple. It is single or multiple, and is usually situated towards the base of the bladder, not far from the neck, so as sometimes to obstruct the channel during micturition. The vessels and the stroma being alike delicate and fragile, the tumour is very apt to bleed, and may thus prove highly dangerous to the patient. From time to time fragments of the villi (Fig. 487) are detached and passed with the urine. The growth is properly speaking not a 'cancer' at all.

Primary **carcinoma** of the bladder is not very common: it takes the form of a smooth infiltrating or nodular and papillary growth, at times spreading over a considerable part of the bladder and penetrating the submucous and even the muscular coat. The cancerous infiltration may thence extend to neighbouring parts. Secondary carcinoma of the bladder arises by the extension of cancerous infiltration from the uterus, vagina, rectum, or prostate.

Other neoplasms of the bladder are very rare indeed; but cases of mucous polypus, myoma, fibromyoma, myxoma, sarcoma, fibrosarcoma, angioma, adenoma, and dermoid tumours are on record: they either form nodular circumscribed growths, or (as in the case of sarcoma) infiltrate the wall of the bladder.

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303. **Dilatation** of the bladder takes place when its evacuation is interfered with through occlusion or stricture of the urethra or paralysis of the muscular wall of the bladder itself. When the evacuation is rendered difficult, or when frequent contraction of the bladder is induced by the stimulus of a stone, the muscular coat may undergo **hypertrophy** (Fig. 490 *d*). The wall becomes thickened, and the overgrown muscle-bundles stand out from the inner surface in a reticulum of ridges or fasciculi (fasciculated bladder).

Diverticula are produced either by simultaneous local yielding of all the coats, or by the protrusion of the mucosa and submucosa through one of the meshes of the fasciculated muscular coat. These diverticula are seldom larger than a walnut. They are frequently the seat of concretions, and sometimes are first caused by the pressure of a vesical calculus.

Displacements of the bladder are rare, though occasionally a portion of the viscus prolapses into a hernial sac. The base of the bladder in women is liable to fall down into the vagina (vaginal cystocele), or the posterior wall may prolapse through the dilated female urethra and appear at the external orifice.

Rupture of the vesical wall results from traumatic injury, excessive distension of the bladder, or morbid change in the wall. Rupture into the abdominal cavity usually leads to fatal peritonitis. After perforation into the pelvic cellular tissue urinary infiltration takes place, leading to suppuration or gangrene in the tissue invaded. Ulceration or local necrosis sometimes leads to the opening of abnormal communications between the bladder and the vagina, uterus, or external cutaneous surface. These are called **urinary fistulae,** and are kept open by the constant escape of urine through them.

CHAPTER C

THE URETHRA

304. The **inflammations** of the urethra correspond generally to those of other mucous membranes. Croupous and diphtheritic inflammations are rare, but catarrh is very frequently met with. The most important form of catarrh, in women as well as in men, is **gonorrhoea**, set up by the specific *Gonococcus* of NEISSER. The micrococcus is conveyed to the urethra in the secretion from another mucous membrane affected with gonorrhoea, and as it multiplies induces an inflammation characterised by its purulent catarrhal exudation, which is yellowish or greenish-yellow and sometimes slightly stained with blood. The inflammation may extend from the urethra to other parts of the urinary tract and to the neighbouring genital organs. In the inflamed region (Fig. 491 *d e*) the epithelium desquamates and leucocytes migrate to the surface, while the fibrous submucous structures are more or less infiltrated with cells and hyperplastic (*b c*).

The inflammation usually ends in recovery, though in places it may lead to ulceration and abscess, to hyperplasia of the connective tissue, to corrugation and thickening of the mucous membrane, or to cicatricial contraction and stricture. These changes are most common in chronic cases (**gleet**, *goutte militaire*). In the male the inflammation persists longest in the membranous portion of the urethra.

Other forms of urethral inflammation are the soft chancre or **chancroid** (Art. 156) and the **hard chancre** or initial sclerosis of syphilis (Art. 159). Ulceration is frequent behind the site of a stricture, and it readily extends to the urethra from prostatic ulcers. When the ulceration goes deeply fistulous tracks may be formed, leading to urinary infiltration of the surrounding tissue and ultimately to abscesses and urinary fistulae.

A not uncommon after-effect of chronic inflammation is the formation of polypous and papillary growths, such as the **cauliflower excrescences** (*condylomata acuminata*) or caruncles that appear round the urethral orifice in women.

Varices resembling rectal haemorrhoids are sometimes formed at the site last-named, in consequence of long-continued inflammatory hyperaemia.

The most common **tumours** affecting the female urethra are sarcoma, myxoma, fibroma, and carcinoma. Fibroma sometimes gives rise to nodose or to vascular papillomatous growths. In male patients cancer of the prostate or of the glans penis frequently attacks the urethra. Small **cysts** of retention are occasionally formed in the mucous glands of the female urethra.

Stricture of the urethra is proximately due to inflammatory swelling of the mucous membrane, to nodular or diffuse unilateral or encircling fibrous hyperplasia, to cicatrices, to valvular folds of membrane, or to polypous growths. Gonorrhoeal inflammation and traumatic injury are the most frequent exciting causes. Inflammatory strictures are oftenest seated in the membranous part of the canal and at the commencement of the spongy portion. In old men the **enlarged prostate** (Fig. 490 *e*) frequently obstructs and even occludes the urethra.

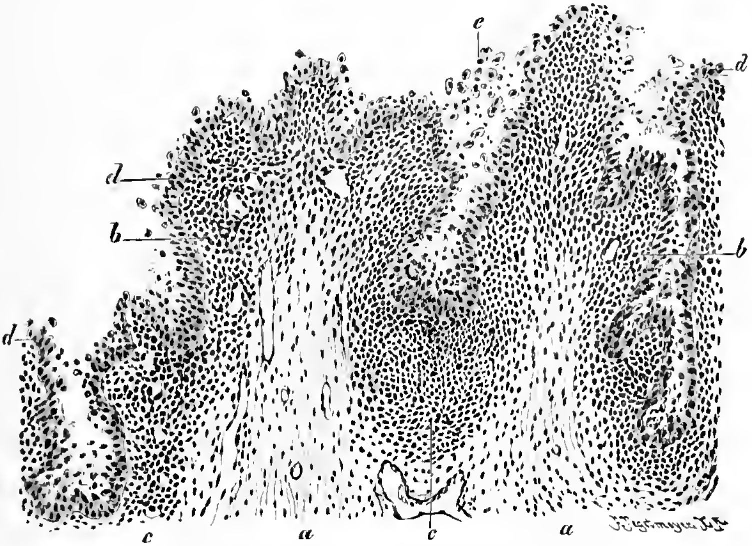

FIG. 491. GONORRHOEAL URETHRITIS.

(*Transverse section through the corrugated mucous membrane: preparation hardened in Müller's fluid, and stained with eosin and haematoxylin:* × 400)

a	normal connective tissue
b c	inflamed, infiltrated, and hyperplastic connective tissue of the mucous membrane
d	infiltrated and desquamating epithelium
e	desquamated epithelium and pus-cells

Traumatic rupture of the urethra arises in various ways; a very common cause is careless catheterisation, by which 'false passages' are produced. They are usually situate at the deeper end of the canal, and either end blindly or lead into the urethra again or into the bladder.

Such ruptures give rise to urinary infiltration and abscess, or to fistulae surrounded by dense fibrous tissue and partially lined with epithelium.

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CHAPTER CI

THE SUPRARENALS

305. Among the **anomalies** of development of the suprarenal bodies the most important are hypoplasia and agenesis, which are met with chiefly in anencephalous and hemicephalous foetuses. According to ZANDER, the development of the suprarenals is imperfect when the anterior part of the cerebral hemispheres is absent, the posterior portions and the base of the brain having apparently no relation to these organs.

Accessory suprarenals are not very infrequent: they lie either close to the main bodies, or remote from them in the kidneys, the liver, or about the genital organs, for example in the broad ligament of the uterus.

Fatty degeneration is a normal phenomenon in the adult: it is apparent chiefly in the cells of the cortical layers, which thereby acquire a pale-yellow tint.

Amyloid degeneration of the blood-vessels is not infrequent as an accompaniment of amyloid disease in other organs: it gives rise to induration.

Pigmentation is of very common occurrence in old age, affecting chiefly the deeper layers of the cortex. The cells are either of a uniform yellow tint or beset with pigment-granules.

Haemorrhage into the suprarenals is somewhat uncommon, though cases occur in which the extravasation is so great as to cause the organ to swell enormously. It is generally due to mechanical injury or to disorder of the circulation, and may lead to the formation of cysts.

Inflammation of the suprarenals is not frequently observed, though it does occur in various forms. Thus in acquired and in hereditary syphilis cellular infiltration and gummatous inflammations are described. And in other cases inflammation ending in suppuration or in cicatricial induration has been noted.

The commonest as well as the most important variety of inflammation is that which terminates in **caseo-fibroid degeneration** of the gland: probably in almost all cases it is of a tuberculous nature. The suprarenals are more or less enlarged, and the capsule is thickened and adherent to the neighbouring structures. The surface is either smooth or nodular and misshapen: on section the parenchyma appears in great part replaced by dense fibrous tissue enclosing caseous patches of various sizes, or collections of cheesy pus. The morbid change is usually bilateral, and leads to the affection known as **Addison's disease** (*morbus Addisonii*), which is dealt with in the volume on General Pathological Anatomy.

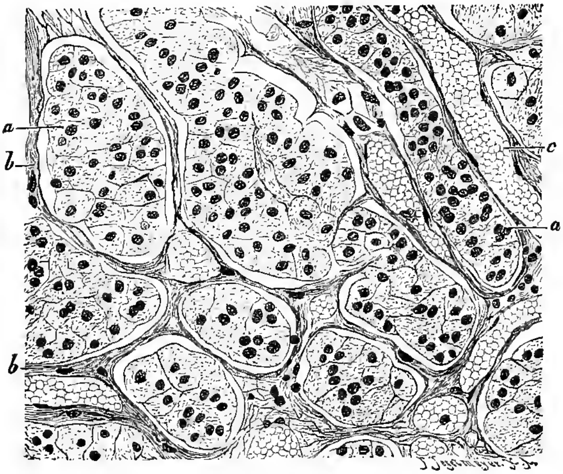

FIG. 492. SUPRARENAL ADENOMA.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin:* × 300)

a groups of epithelial cells *b* stroma
c blood-vessel

The **tumour** oftenest observed in the suprarenals is that described by VIRCHOW, as *struma lipomatosa suprarenalis* or **adenoma** (Fig. 492), a hyperplastic overgrowth consisting essentially of fatty glandular tissue, which leads to considerable enlargement of the organ. Carcinoma and melanotic (DOEDERLEIN, KUSSMAUL) and non-pigmented sarcoma also occur, the latter sometimes attaining a very large size. According to WEICHSELBAUM and DAGONET tumours containing ganglion-cells and nerve-fibres are sometimes found in the suprarenals.

Post-mortem softening of the central portions of the organ is not uncommon, and gives rise to the appearance of morbid excavation.

The *Echinococcus* is the only animal parasite met with in the suprarenals.

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SECTION XIII

THE GENITAL SYSTEM

CHAPTER CII

THE TESTIS AND ITS APPENDAGES

306. The **testis** is a compound tubular gland made up of convoluted and anastomosing tubules arranged in small conical lobules. Externally the organ is enveloped in a dense fibrous capsule, the *tunica albuginea*, whose outer surface is lined by the serous *tunica vaginalis*. From the tunica albuginea somewhat stout fibrous septa pass inwards between the lobules, separating them from each other, and converging to a posterior longitudinal ridge of fibrous tissue known as the *mediastinum testis* or *corpus Highmori*. This mediastinal ridge is traversed by the seminal ducts of the lobules, which enter it as straight narrow tubules, and in its substance break up into a network of ramifying canals known as the *rete vasculosum testis* or *rete Halleri*. The seminiferous tubules themselves are comparatively wide, and are lined by several layers of epithelial cells resting on a stout basement-membrane. After puberty certain of these cells are transformed into spermatozoa. The intertubular stroma is a loose fibrous tissue abounding in blood-vessels, lymphatics, and cells. The straight tubules are narrower than the seminiferous tubules, and are lined with low columnar epithelial cells seated on a delicate basement-membrane. The canals composing the rete have no proper walls, but are merely a series of anastomosing channels in the fibrous stroma, lined with small flattened cells.

The **epididymis** lies upon the corpus Highmori and is composed of a head or *globus major* and a tail or *globus minor*: it receives the *vasa efferentia* which are continuous with the channels of the rete testis: when they reach the head of the epididymis after many complicated convolutions their coils are grouped in a series of small conical lobules known as *coni vasculosi*. The several tubules of the coni vasculosi converge and empty into one larger duct or canal, the manifold subsequent convolutions of which make up the head and tail of the epididymis. At the tail of the epididymis the canal comes to an end, and its channel becomes continuous with that of the *vas deferens* or excretory duct of the testis, which turns up behind the testis along the inner side of the epididymis, beginning with a somewhat sinuous course but becoming straighter as it enters the spermatic cord with which it ascends into the pelvis.

The tubules of the epididymis are lined with a single layer of tall ciliated columnar epithelium, with smaller interposed cells, resting on a thin basement-membrane; they are furnished with circular muscular fibres. The convolutions are held together by somewhat abundant vascular areolar tissue. The free portion of the epididymis is enveloped in a firm fibrous membrane, corresponding to the tunica albuginea, but somewhat finer in texture: the serous tunica vaginalis extends over this, being indeed originally continuous with the abdominal peritoneum.

The epididymis normally lies on the posterior border of the testis, the vas deferens ascending along its inner side.

Complete **absence** of one or both testes is very rare. In such cases the epididymis also is usually lacking, and the vas deferens is rudimentary.

Complete absence or partial defect of the epididymis is very rarely met with when the testis itself is perfectly formed. Partial defect and atresia of the vas deferens occur with or without concomitant malformation of the epididymis.

Cases of congenital **hypoplasia** of the testis with defective development of the seminiferous tubules, and of imperfect evolution of the immature organ at puberty, are not uncommon.

Malposition of the testis is usually due to some interference with the normal descent of the organ. Such an abnormality is known as ectopia or retention of the testis, and is distinguished as internal (abdominal) or external. In the former case the testis remains within the abdominal cavity (cryptorchism), and lies either in its initial embryonic position (lumbar abdominal ectopia), or near the internal opening of the inguinal canal (iliac abdominal ectopia). Where the testis lies outside the abdominal cavity, the ectopia is termed inguinal if the organ is lodged in the canal, pubic if it is just outside the external ring, cruro-scrotal if it is in the fold between the scrotum and the thigh, perineal if it is nearer the middle line anterior to the anus, and crural if it is in the groin.

An undescended testis which at birth is still in the abdominal cavity or inguinal canal may descend into the scrotum at the time of puberty. The epididymis usually accompanies the misplaced testis, but occasionally the epididymis becomes separated from the latter and enters the scrotum alone. In very rare cases both testes occupy the same pouch of the scrotum (VON LENHOSSÉK).

Malposition of the testis may be unilateral or bilateral, and with it other forms of imperfect development of the genital organs are frequently associated. The misplaced or undescended organ is sometimes defective, or fails to develope at puberty: it often becomes degenerate and atrophic, particularly when it lies in the inguinal canal and is thereby exposed to constant pressure.

Occasionally the testis is misplaced within the scrotum, so that for example the epididymis is in front with its head downward; this condition is described as inversion.

Dislocation of the testis, as distinguished from congenital ectopia, is in rare instances produced by traumatic or other injury, the organ being forced from its proper site into the perineum, the femoral canal, or the inguinal canal.

Atrophy of the testis is commonest as a senile condition, but it also results from mechanical injury, inflammatory disease (Art. 307), and the invasion of new-growths, as also from certain affections of the central nervous system. The use of iodine is said to be capable of causing atrophy of the organ.

In an atrophic testis no spermatozoa are produced. The seminiferous tubules contain colourless granules, fat-droplets, and pigment-flakes: the tissue of the organ appears pale and flabby. As the testis wastes the epididymis sometimes remains unchanged; in other cases however it also undergoes atrophy.

References on the Morbid Anatomy of the Testis.

Arthaud: *Étude sur le testicule sénile* Paris **1885**
Bramann: The processus vaginalis in cases of retained testis *A. f. klin. Chir.* XL **1890**
Brazzolo: Normal and morbid histology of the testis *Mem. d. R. accad. d. scienze di Bologna* IX **1888**
Curling: *Observations on the structure and diseases of the testis* London **1886**
Ebner: *Untersuch. über den Bau der Samenkanälchen* Leipzig **1871**
Englisch: *Art.* Testis *Eulenburg's Realencyklop.* IX **1887**
Griffini: Partial reproduction of the testis *A. per le scienze med.* XI **1887**
Griffiths, J.: Structural changes in aged and in eunuchoid persons *Journ. of Anat.* XXVII **1893** and XXVIII **1894**
Gruber: Cryptorchism *V. A.* 73 **1878**
Kocher: *Die Krankheiten der männlichen Geschlechtsorgane* Stuttgart **1887**
von Lenhossék: Ectopia testis transversa *Anat. Anzeiger* I **1886**
Lesser: *Geschlechtskrankheiten* Leipzig **1890**
Lockwood: Anomalies of development and transition *Journ. of Anat.* XXI **1887** and XXII **1888**
Monod and Terrillon: *Traité des maladies du testicule* Paris **1889** (with references)
Ribbert: Compensatory hypertrophy of the sexual glands *V. A.* 120 **1890**
Riese: Pathology of the male urogenital system *Cent. f. allg. Path.* III **1892**
Stilling: Experiments on the atrophy of misplaced testes *Ziegler's Beiträge* XV **1894**
Weil: *Ueber den Descensus testiculorum* Prague **1885**

307. **Orchitis**, or inflammation of the testis, and **epididymitis**, or inflammation of the epididymis, are induced by traumatic injury, by haematogenous infection, or by the direct extension of inflammatory affections of the bladder or urethra along the vas deferens.

Traumatic and transmitted inflammations are commoner in the epididymis than in the testis. Haematogenous inflammations chiefly affect the testis; they make their appearance mainly in connexion with pyaemia, epidemic parotitis or mumps, small-pox, scarlatina, typhoid fever, and syphilis. Other infective diseases also are occasionally the forerunners of secondary orchitis and epididymitis.

In small-pox, according to CHIARI, small patches of cellular infiltration make their appearance in the testicular tissue in a considerable proportion of cases: the patches sometimes attain the size of a pea, and within them the epithelial structures become necrotic. As they heal they undergo cicatrisation.

Among the irritants that reach the epididymis from the urethra by way of the vas deferens, and induce epididymitis (*epididymitis urethralis*), the most important are the *Gonococcus* and the tubercle-bacillus; but those contained in the urine in cases of purulent, croupous, or gangrenous cystitis, urethritis, or prostatitis occasionally set up both epididymitis and orchitis. The inflammatory changes following operations on the bladder or urethra at times extend to the epididymis along the vas deferens.

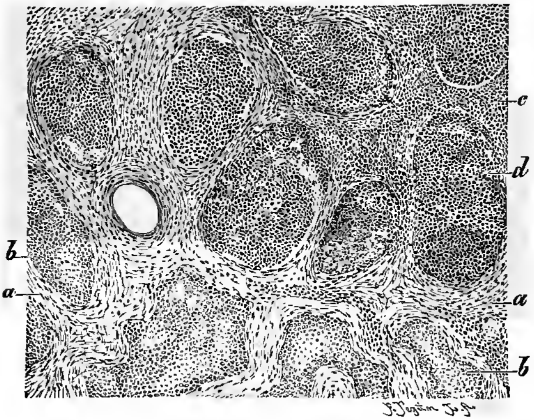

FIG. 493. PURULENT ORCHITIS.

(*Consecutive to purulent paralytic cystitis and epididymitis: preparation hardened in Müller's fluid, and stained with haematoxylin*: × 45)

a fibrous stroma
b seminiferous tubules
c infiltrated stroma
d tubules distended with pus

In orchitis the inflammatory exudation collects both in the intertubular stroma and in the seminiferous tubules, and gives rise to very considerable swelling of the testis. When the inflammation issues in local suppuration, great masses of leucocytes accumulate in the tubules and stroma (Fig. 493 *c d*).

The epididymis exhibits similar changes (Fig. 494). In gonorrhoeal epididymitis, for example, the epithelium lining the tubules is sometimes densely infiltrated (*a*), and their channels are distended, with round-cells. The epithelium also becomes mucoid and is desquamated (*b*), and the appearances are thus not unlike those of catarrhal inflammation affecting a mucous membrane.

In traumatic inflammation the process is often complicated by the extravasation into the tissues of blood from the lacerated vessels. As recovery takes place the extravasated blood and the inflammatory exudation are generally re-absorbed, the lost epithelium being made good by regenerative multiplication of the remainder. Sometimes however the tissue of the testis and epididymis is permanently damaged and never regains its normal appearance.

When the inflammation assumes a purulent character the tissue sometimes breaks down by suppuration, and an **abscess** is produced, as in cases of metastatic pyaemia and of urethral infection in connexion with gonorrhoea, and after lithotripsis and urethrotomy or other operation for urethral stricture. Such abscesses are at times no larger than a pea and lie embedded in the substance of the organ, in other cases they are as large as a chestnut, and protrude from the surface or give rise to general enlargement. Very small abscesses generally undergo absorption and become cicatrised: the larger accumulations of pus keep up a constant irritation, and lead to the development of granulations and of new fibrous tissue, which gradually encapsules the pus and permits it to become inspissated into a semi-solid pulp consisting of fatty detritus and cholesterin. Sometimes the encapsuling membrane itself secretes fresh pus, and the abscess again enlarges. Usually the abscess is single, but now and again a number of them are formed simultaneously in the same testis.

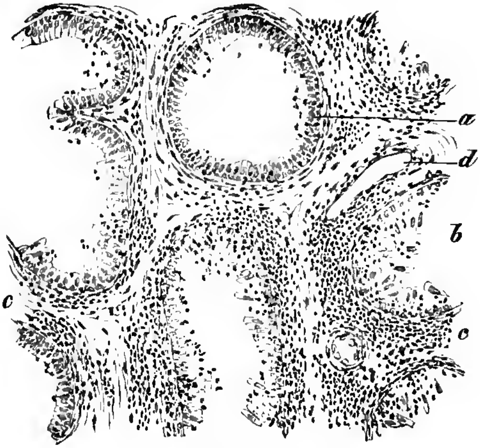

Fig. 494. Epididymitis.

(Preparation hardened in alcohol, and stained with haematoxylin: × 40)

a tubule of the epididymis whose epithelium retains its form but is infiltrated with round-cells
b tubule whose epithelium is densely infiltrated with round-cells and in process of desquamation
c intertubular connective tissue infiltrated with cells

As a rule the tunica vaginalis is not involved if the abscesses are very small and deeply-seated; but in many cases the suppurative inflammation extends to the serous surface, and results in the effusion of serous, sero-fibrinous, or purulent exudations into the vaginal sac between the testicular and the scrotal layers of the membrane. Sometimes the abscess breaks through the tunica, and the external coverings of the testis and finally the skin of the scrotum are thereby involved in the suppurative process. The scrotum itself may thus be perforated, and the exposed and partially-protruding [illegible] vered with granulations (non-malignant fungus), which project more or less luxuriantly from the opening in the skin.

In the indurated tissue surrounding a chronic abscess the seminiferous tubules are either obliterated or in some degree atrophic. As the result of epididymitis the vas deferens is occasionally obliterated, while other portions of the system of canals are distended into cysts by the retained secretion.

The result of cicatrisation in foci of degeneration or inflammatory infiltration, from whatever cause arising, is always atrophy of the glandular structures and hyperplasia of the fibrous stroma, or in other words the formation of patches of functionless and indurated tissue. These when the active changes are at an end appear as whitish scars and fibrous bands pervading the testis or epididymis as the case may be.

Within such cicatricial patches in the testis the seminiferous tubules are often entirely obliterated, while at their margins the connective-tissue elements of the tubular walls are usually thickened (Fig. 495 *a*), the intertubular fibrous stroma is widened, and the epithelium has entirely disappeared (*a*) or is at least atrophic. These white atrophic patches are not infrequently met with in the senile testis, but the primary or exciting cause of the morbid process of which they are the result cannot in general be made out with certainty. When epididymitis results in induration (Fig. 496) the distorted and displaced tubules sometimes take the form of peculiar cellular clusters and columns (*d*) resembling in appearance the cell-nests and claw-like processes of cancer. To judge from the microscopical appearances, the proliferation of the epithelial cells sometimes assumes a non-typical character, and this gives rise to still greater diversity in the forms of the cellular aggregations. In certain cases the epithelium even secretes mucus, with the result that small mucous cysts are formed, and some of the tubular channels, being distended with fatty desquamated

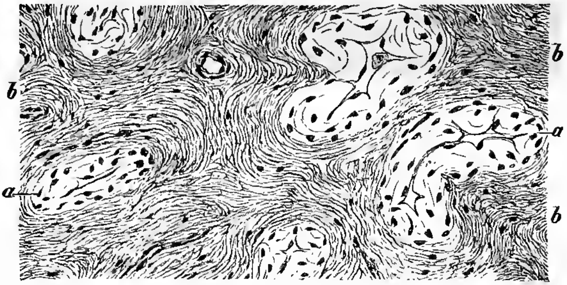

FIG. 495. ATROPHIC TESTIS WITH LUSTROUS WHITE PATCHES OF LOOSE CICATRICIAL TISSUE.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin* : × 60)

a thickened wall of atrophic tubules *b* hyperplastic intertubular stroma epithelial cells and with fatty immigrant leucocytes, are converted into solid whitish-looking cords.

Throughout the course of the inflammation the fibrous stroma of the testis and of the epididymis (*c*) is infiltrated with cells. When it subsides the cells appear less numerous, and the tissue generally becomes more compact (*a*) or even sclerotic, though parts of it may undergo a kind of myxomatous metaplasia (*b*).

When the scrotum and tunica vaginalis are wounded and the testis thereby exposed, granulations usually make their appearance on the surface of the tunica albuginea, and form a fungating excrescence protruding through the opening in the skin; this, as we have already stated, is known as benign or **non-malignant fungus** of the testis. If the tunica albuginea also is lacerated, some of the seminiferous tubules are apt to be forced through the opening and so destroyed, while granulations presently spring up over the damaged portion of the testis. As the scrotal wound closes by cicatrisation the exuberant granulations cease to be formed, and the process terminates by the production of a scar.

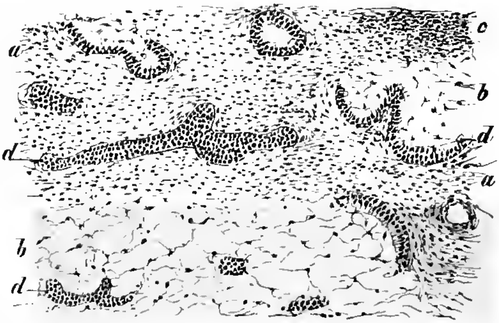

FIG. 496. ATROPHIC DEGENERATION OF THE EPIDIDYMIS FROM TRAUMATIC EPIDIDYMITIS.

(*The traumatic injury was received twelve years previously, and was followed by epididymitis, haemorrhagic periorchitis, and hydrocele of the cord: preparation hardened in Müller's fluid, stained with alum-carmine, and mounted in Canada balsam:* × 40)

a compact fibro-cellular tissue
b mucoid tissue
c cellular infiltration
d remains of the tubules of the epididymis

References on Epididymitis and Orchitis (see also Art. 306).

CHIARI: Orchitis in small-pox *Prager Z. f. Heilk.* VII **1886** and X **1889**
FANEUIL: Orchitis in small-pox *Thèse* Paris **1873**
FINGER: *Die Blennorrhöe der Sexualorgane* Leipzig **1896**
HAMILTON: Acute orchitis *Practitioner* XXXI London **1883**
HARDY: Studies on the inflammations of the testis *Thèse* Paris **1860**
JACOBSON: Orchitis *V. A.* 75 **1879**
LE DENTU: Malarial orchitis *Bullet. Soc. de chir.* XIII Paris **1887**
MONOD and TERRILLON: *Traité des maladies du testicule et de ses annexes* Paris **1889**
RIGAL: Experiments on traumatic orchitis *A. de physiol.* VI **1879**
SHEILD: Suppurative affections *Med.-chir. Trans.* LXXIV London **1890**
STEINER: Chronic orchitis *A. f. klin. Chir.* XVI **1873**
TAVEL: Orchitis in typhoid fever *Correspbl. f. Schweizer Aerzte* **1887**
TÉDENAT: Orchitis following mumps *Montpellier méd.* **1884**, and *Mémoires de chirurgie* Montpellier **1872**
ZACCO: Malarial orchitis [illegible] **1884**

308. **Tuberculosis** of the testis and epididymis (Fig. 497) is a somewhat common affection: it usually begins in the epididymis, rarely in the testis, and is met with in children as well as in adults.

The tuberculous lesion in these organs may be the only one in the body, but in general other organs are simultaneously affected. Sometimes the tuberculous orchitis is only one manifestation of a general urogenital tuberculosis, involving in particular the prostate, the seminal vesicles, and the bladder.

The tubercles are not usually disseminated and uniform in appearance; much more frequently when the organ comes under examination it is occupied by one or more large caseous nodes (Fig. 497 b_1) together with numerous smaller nodules (a). The epididymis (b b_1) is commonly enlarged, and is either more or less completely converted into a caseous mass enveloped in a fibrous capsule, or uniformly indurated and studded with a few caseous nodes whose centres are softened and broken down.

Next to the epididymis the corpus Highmori is the part that is most apt to be diseased, its substance being studded with cheesy nodes or entirely caseous, while the parenchyma of the testis (a) contains only a few small grey or yellow tubercles. The latter may however contain some larger caseous patches surrounded either by a greyish semi-translucent zone or by indurated fibrous tissue. At times the greater part of the testis is destroyed, its substance being pervaded by softened or still firm caseous deposits, or almost entirely transformed into a continuous caseous mass whose centre has undergone softening.

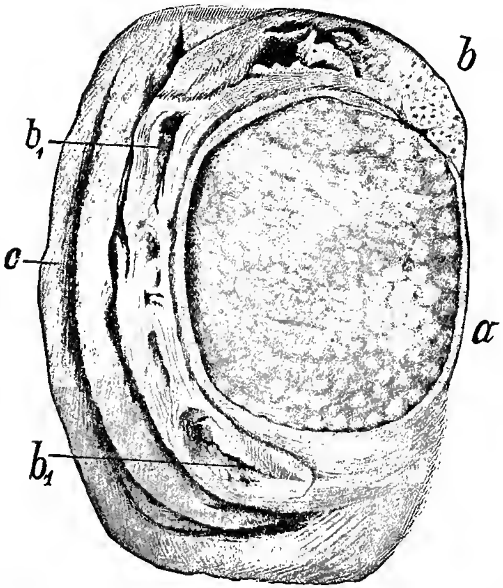

FIG. 497. TUBERCULOSIS OF THE TESTIS AND EPIDIDYMIS.

(*Natural size*)

a testis studded with tubercles
b epididymis with softened caseous nodes b_1
c skin

In addition to the caseous tubercles seated in the stroma, soft pulpy caseous deposits are met with in the dilated tubules of the organ, and particularly in those of the epididymis.

From a tuberculous lesion in the epididymis or testis the specific infection may be conveyed to other parts not only by way of the lymphatics but also through the seminal canals. The lymphatic mode of diffusion gives rise first to granulomatous infiltration of the intertubular stroma, which then extends to the contiguous tubules: but when the bacilli are conveyed by the seminal channels, the walls of the tubules are first invaded, and when they are broken through the surrounding structures become consecutively involved. The specific tuberculous process is not infrequently accompanied by wide-spread catarrh of the seminal tract.

In many cases the affection is limited to the testis or epididymis; but occasionally it extends to the serous surface, and invading both layers of the tunica vaginalis finally involves the skin of the scrotum. Such extension into the external coverings takes place with special facility from the corpus Highmori and the epididymis.

The route followed by the disease is marked by the appearance of nodules, granulations, cheesy deposits, and softened patches. When tubercles form on the tunica vaginalis, serous or sero-fibrinous exudations are effused into the sac of the serous membrane. Sooner or later the subcutaneous foci break through the external coverings, and fistulous tracts and ulcers lined with tuberculous granulations are produced. The partially-denuded and exposed testis also becomes covered with fungating granulations, and protrudes through the ulcerous opening in the scrotum as an exuberant mass varying from the size of a hazel-nut to that of a hen's egg; this condition is described as **tuberculous fungus** of the testis. Beneath the granulations the parenchyma of the testis and epididymis is infiltrated with cells and studded with tubercles. The tunica albuginea sometimes preserves its integrity, but in other cases it is destroyed entirely or in part, and the tuberculous granulations then spring directly from the glandular parenchyma.

Syphilitic orchitis is not infrequent in the later stages of syphilis: it gives rise to intertubular infiltration combined with swelling of the testis. It ends either in fibrous induration of the part, accompanied by atrophy of the glandular tissue (Art. 307, Fig. 495), or in the formation of firm caseous gummatous nodes surrounded by cicatricial tissue. In congenital syphilis also enlargement and induration of the testis are occasionally though not very frequently induced, the morbid change being due to cellular infiltration and fibrous hyperplasia.

In simple induration the parenchyma of the testis is traversed by strands of white fibrous tissue running chiefly from the mediastinum to the tunica albuginea. When gummata are present the indurated tissue usually encloses one or more caseous patches.

The hyperplastic new tissue is at first highly cellular, but afterwards it becomes more purely fibrous and sclerotic, while the glandular tissue it encloses undergoes atrophy or disappears entirely. The coats of the arterioles, and in particular the intima, are greatly thickened.

In syphilis the epididymis is very seldom primarily affected, but it is often enough involved by extension of the disease from the testis. The tunica albuginea also is nearly always invaded by the specific inflammation, which gives rise to serous or sero-fibrinous exudation into the vaginal sac, or to fibroid induration and adhesions between the apposed surfaces. The parietal layer of the tunica vaginalis and ultimately the skin of the scrotum are sometimes successively involved, and softened gummata may even break through to the exterior. When the testis protrudes through the opening and becomes covered over with granulations, the result is spoken of as **syphilitic fungus**, but this result is much less common than in tuberculosis.

In **leprosy** we sometimes meet with inflammatory nodes and thickenings in the testis and epididymis, the glandular tissue of which is thereby destroyed. Sometimes the nodes appear to be re-absorbed, but the atrophic condition of the testis remains.

References on Tuberculosis and Syphilis of the Testis and Epididymis (see also Art. 306).

Balme: Syphilitic epididymitis *Thèse* Paris **1876**
Curling: *Diseases of the testis* London **1878**
Demme: Tuberculosis of the testis *V. A.* 22 **1861**
Dron: Syphilitic epididymitis *A. gén. de méd.* II **1863**
Fournier: *Du sarcocèle syphilitique* Paris **1875**
Gaule: Tuberculosis of the testis *V. A.* 63 **1875** and 69 **1877**
Heiberg: Primary urogenital tuberculosis *Virchow's Festschrift (internationale)* II Berlin **1891**
Henoch: Syphilitic orchitis in new-born infants *D. Z. f. prakt. Med.* **1877**
Hutinel: Syphilitic orchitis in new-born infants *Rev. mensuelle de méd.* **1878**
Lübimow: Histogenesis of giant-cells *V. A.* 75 **1879**
Malassez: Tuberculous granulations of the testis *A. de physiol.* III **1876**
Malassez and Réclus: Syphilis of the testis *A. de physiol.* VIII **1881**
Réclus: *Du tubercule du testicule etc.* Versailles **1876**; *De la syphilis du testicule* Paris **1882**, and *Gaz. hebdom.* **1883**
Simonds: Tuberculosis *D. Z. f. Chir.* XVIII **1882**
Tédenat: Syphilitic affections of the testis *Montpellier méd.* **1878**
Virchow: *Die krankhaften Geschwülste* II
Waldstein: Tuberculous affections of the testis *V. A.* 85 **1881**

309. **Cysts** (Fig. 498 *c*) due to distension of the tubules of the testis and epididymis by retained secretion are most frequently met with at the junction of the rete vasculosum and the vasa efferentia, or near the head of the epididymis; they rarely occur elsewhere in the tubular system. The contents of these cysts are clear or milky (galactocele) and are often mingled with spermatozoa (spermatocele). The cyst-wall is lined either with tall columnar epithelium, which is often ciliated, or with low cubical and flattened epithelium.

Certain cysts of the head of the epididymis, generally found in old patients, continue small and have no appreciable importance. Those appearing in earlier life (Fig. 498 *c*) are of graver significance: they gradually increase in size, and occasionally contain as much as from fifty to one hundred cubic centimetres of liquid. They are either due to antecedent inflammation leading to dilatation of the vasa efferentia, or arise idiopathically in the *vasa aberrantia*, or diverticula from the canal of the epididymis that are either abstricted and so closed or communicate freely with the seminal channels. Such aberrant cysts occur in the rete vasculosum (M. ROTH) as well as in the epididymis. It is not unlikely that certain small cysts lined with ciliated epithelium occasionally observed in the testis are also due to dilatation of remnants of the foetal (wolffian) ducts.

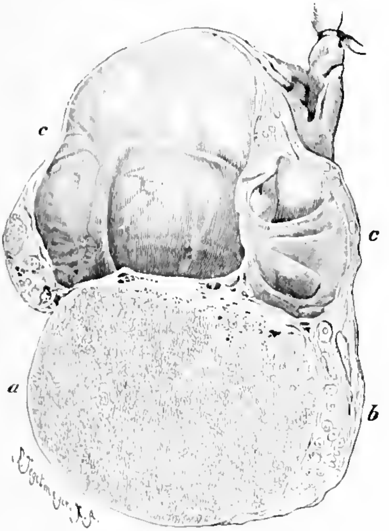

FIG. 498. CYSTS OF RETENTION IN THE EPIDIDYMIS.
(*Natural size*)
a testis *b* epididymis *c* cysts

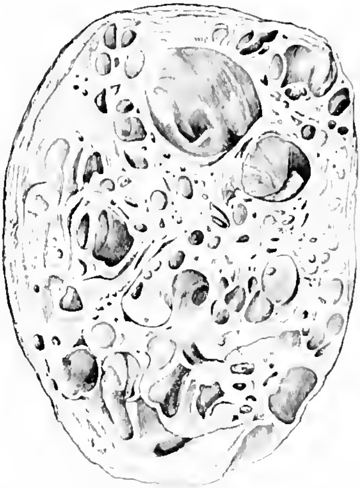

FIG. 499. CYSTADENOMA OF THE TESTIS.
(*Section of the testis of a boy aged four years: natural size*)

Small cysts lie hidden in the substance of the gland or project slightly above its surface. Larger cysts force asunder the epididymis and the testis, produce general enlargement of the latter, or protrude from its surface. When such a cyst contains spermatozoa, its cavity must of course at one time or another be in communication with one of the seminal channels. According to ROTH, spermatocele of the hydatid of Morgagni (first described by LUSCHKA) is due to the gradual dilatation by seminal liquid of an aberrant diverticulum of the canal of the epididymis which ends blindly in the hydatid.

Of the **tumours** of the testis and epididymis the first to be described are **adenoma** and **cystadenoma** (Fig. 499). They are found both in children and in adults, some of them at least being undoubtedly attributable to disorders of development. They sometimes form tumours of considerable size, especially when much liquid gathers in the tubular canals. Their contents consist of a clear or blood-tinged slimy liquid, or a yellowish-white greasy pulp like that contained in a cutaneous wen (atheroma): we may distinguish these varieties as mucous and atheromatous cystadenoma respectively. In the former the cyst-wall is lined internally with one or more layers of columnar epithelial cells, which are sometimes ciliated, and the contents are mainly due to the mucoid degeneration of these cells. The atheromatous cysts are lined with stratified epithelium resembling the rete Malpighii of the skin, and the contents consist of fatty detritus and epithelial scales, with epithelial 'pearls' in some cases. These tumours are usually seated in the testis; but they occasionally invade the epididymis by extension or arise within it independently. Circumscribed patches of cartilaginous tissue (Fig. 500 *e*) are often observed in the substance of the growths, giving them the character of **chondro-adenoma** or **chondrocystoma**. Free proliferation of the stroma transforms them into the varieties known as **adenosarcoma** or **cystosarcoma**. Cystadenoma on the other hand sometimes undergoes a kind of carcinomatous metaplasia, or is combined with actual carcinoma: thus, for example, the testis may be the seat of a typical cancer (Fig. 500 *f*) while at the same time a cystadenoma or chondrocystoma (d d_1 e) developes in the epididymis. In certain very rare cases striated muscular fibres have been seen in the stroma of cystadenomata (BILLROTH, SENFTLEBEN).

Carcinoma (Fig. 500 *f*) is one of the commonest tumours of the testis: it takes the form either of soft marrowy growths (encephaloid or medullary carcinoma), or of firmer and more compact tumours with an abundant stroma (simple and scirrhous carcinoma). It often happens that different parts of the same growth differ in structure. According to LANGHANS cancer of the testis usually starts in the convoluted seminiferous tubules. The cancer-cells are exceptionally prone to mucoid and fatty degeneration, and in the softer forms haemorrhage is very common; sections of such growths are thus apt to be mottled with various tints.

Cysts with gelatinous or colloid contents are produced by the mucoid or colloid degeneration of the cells in the cancerous loculi, the tumour being then described as cystocarcinoma or colloid carcinoma.

Carcinomata likewise in many cases contain rounded nodes and nodules or elongated, ramified, and cactus-like patches of cartilaginous tissue, chiefly in the neighbourhood of the rete testis and epididymis. These patches as they grow sometimes break into the lymphatics and seminal canals of the testis and therein ramify, assuming the most diverse forms. In this case we might distinguish the tumour as **chondrocarcinoma**.

In cases of carcinoma of the testis the fibrous stroma of the growth itself or that of the epididymis sometimes undergoes sarcomatous proliferation. Metastasis takes place both through the blood-vessels and through the lymphatics.

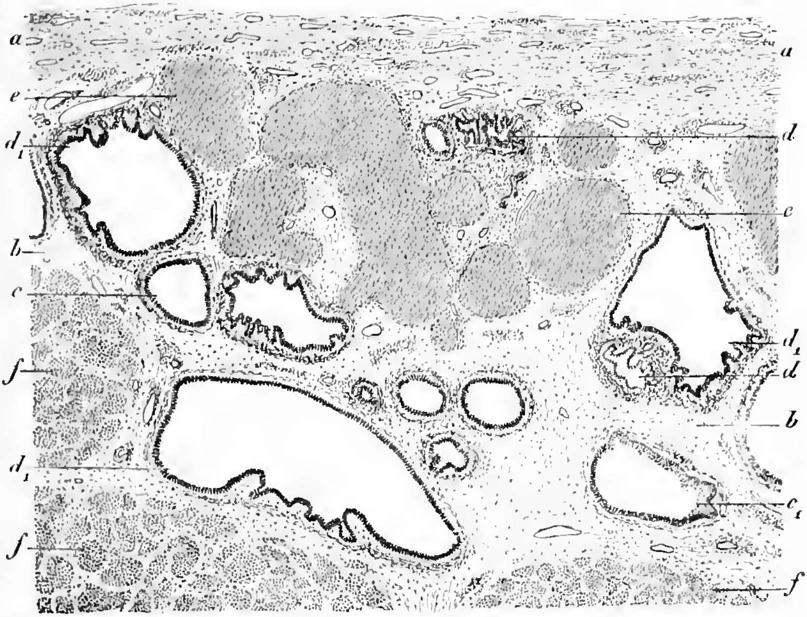

FIG. 500. CARCINOMA OF THE TESTIS WITH CARTILAGINOUS PATCHES AND PROLIFEROUS CYSTS IN THE EPIDIDYMIS.

(Section from the external margin of the tumour: hardened in Müller's fluid, stained with alum-carmine and neutral ammonium carminate, and mounted in Canada balsam; × 8)

a	external covering of the tumour corresponding to the tunica albuginea of the epididymis
b	stroma of the epididymis
c c_1	cystic tubules of the epididymis
d d_1	tubules of the epididymis containing papillary outgrowths from their walls
e	cartilaginous patch
f	nests of cancer-cells

Simple **enchondroma** of the testis is very rare. It generally originates in the rete vasculosum (KOCHER) or in the epididymis, and consists of one or more nodes which may be as large as a walnut, or of a great number of smaller cartilaginous patches like those just described, embedded in the fibrous stroma.

Fibromata have been seen in a few cases in the rete and tunica albuginea, in the form of little nodes, some of them calcified. ROKITANSKY, NEUMANN, and ARNOLD have described fleshy tumours chiefly composed of thick and thin fibres, some of which were longitudinally and others transversely striated: these growths must accordingly be classed as examples of striocellular myoma or **rhabdomyoma.** In the case described by Arnold the parenchyma of the testis was entirely replaced by fleshy masses; while in the cases reported by Rokitansky and Neumann the tumour was situated in the lower end of the organ.

Myxomata are likewise rare; but the stroma of cystomatous tumours occasionally consists of mucous tissue, and portions of the substance of a sarcoma sometimes become myxomatous. Similarly osseous and adipose tissue now and then make their appearance in the fibrous stroma of cystadenomata and carcinomata, or form part of the structure of a sarcoma. Pure **osteomata** are very rarely met with (Neumann).

Almost all the forms of **sarcoma** that occur elsewhere are found in the testis, including medullary round-celled sarcoma, lymphosarcoma, alveolar sarcoma, spindle-celled sarcoma, myxosarcoma, fibrosarcoma, giant-celled sarcoma, angiosarcoma, and melanotic sarcoma. It usually originates in the testis, rarely in the epididymis, although the latter is generally invaded by the growth at an early stage. According to its special structure it forms a hard or a soft and marrowy growth, and now and then attains a very considerable size. In some cases the seminiferous tubules involved in the growth undergo cystic dilatation, giving rise to what is termed cystosarcoma. When the sarcomatous tissue invades the wall of the cyst and projects into its cavity in the form of papillary elevations, the tumour is known as a papilliferous or papillomatous cystosarcoma.

Fatty degeneration, caseation, haemorrhage, and softening often take place in sarcomatous tumours, producing a mottled appearance in the cut surface; and they are sometimes excavated by cysts due to local liquefaction of their tissue.

Secondary growths are disseminated by the blood-vessels as well as by the lymphatics, particularly in the case of the medullary variety. It is rare for a sarcoma to break through the tunica albuginea. The growth is commonest in young persons, although it may appear at any age.

Simple and complex **dermoids** are seldom found in the testis.

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Birch-Hirschfeld: Carcinoma *A. d. Heilk.* IX **1868**
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Brousses and Gérardin: *Du lymphadénome* Paris **1886**
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Ehrendörfer: Tumours of the testis *A. f. klin. Chir.* XXVII **1882**

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Griffiths: Cysts of the epididymis and testis *Journ. of Anat.* XXVIII **1894**

Grohe: Spermatocele *V. A.* 32 **1865**

Haward: Cystic disease of the testicle *Trans. Path. Soc.* XXVIII London **1877**

Hochenegg: Cysts in the testis and epididymis *Wien. med. Jahrb.* XV **1885**

von Kahlden: Neoplasms in cryptorchism and monorchism *Münch. med. Woch.* **1887**

Láng: Dermoids *V. A.* 53 **1871**

Langhans: Tumours of the testis *Kocher's Krankheiten des Hodens* Stuttgart **1887**

Luschka: Appendicular structures of the testis *V. A.* 6 **1854**

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Paget: Chondroma (probably carcinomatous) *Med.-chir. Trans.* XXXVIII London **1855** (rediscussed by Kanthack and Pigg: *Trans. Path. Soc.* London **1897**)

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Pietrusky: Carcinoma sarcomatodes *Inaug. Diss.* Greifswald **1889**

Poirier: Pathogenesis of cysts of the epididymis *Rev. de chir.* **1890**; *Verhandl. internat. med. Congress* II Berlin **1891**

Rosenbach: Spermatocele *A. f. klin. Chir.* XIII **1872**

Roth: Formation of cysts and spermatocele *V. A.* 68 **1876** and 81 **1880**

Silcock: Cystic disease *Trans. Path. Soc.* XXXIX London **1887**

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Waldeyer: Various tumours of the testis *V. A.* 44 **1868**; Carcinoma *V. A.* 55 **1872**

310. The **vaginal process** of the peritoneum, which invests the testis as the tunica vaginalis, becomes in normal conditions shut off from the abdomen when the testis descends into the scrotum, and becomes a closed sac; but not infrequently the closure remains incomplete, and then the cavity of the vaginal sac communicates more or less freely with the abdomen, or portions of the intervening canal following the course of the spermatic cord are either abstricted entirely or open at one end into the peritoneal cavity. When the testis occupies some abnormal position outside the abdomen, it is enveloped in a diverticulum of the peritoneum, which is converted into a closed sac or remains in communication with the abdominal cavity.

Inflammation of the tunica vaginalis is the chief morbid affection to which this membrane is liable; it is variously described as **periorchitis** (Kocher), vaginalitis (Vidal, Lancereaux), or vaginitis testis.

Periorchitis is either acute or chronic, and primary or secondary. Primary inflammation may be due to traumatic injury or to other causes which cannot be certainly determined: secondary inflammation is consecutive to orchitis or epididymitis. It is when the functional activity of the testis is at its height that the liability to inflammation is greatest. The affection is more frequent and assumes a graver form in tropical and subtropical countries than in colder climates.

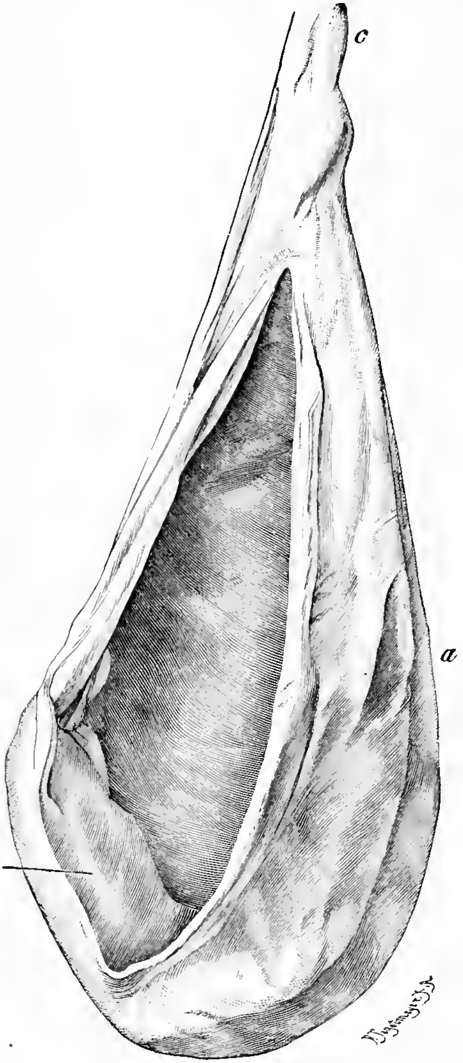

FIG. 501. SEROUS HYDROCELE OF THE VAGINAL SAC.

a sac laid open
b testis flattened by pressure
c spermatic cord

Serous and sero-fibrinous periorchitis gives rise to the collection of serous or sero-fibrinous liquid in the sac of the tunica vaginalis (Fig. 501 *a*). The affection may be acute or chronic: in the latter case it either follows upon an acute attack of the disease or appears gradually and insidiously. As the inflammation continues the liquid increases steadily or *per saltum*, the latter symptom indicating the intercurrence of acute exacerbations. When the quantity of liquid collected in the tunica vaginalis is so large as to be externally perceptible, the condition is usually described as serous **hydrocele** of the vaginal sac (Fig. 501).

In the course of months the quantity of liquid collected in this sac may reach from 500 to 3000 grammes, producing a huge swelling which may distend the scrotum up to the level of Poupart's ligament. The testis is usually situated behind and below the liquid.

At first the effused liquid may contain fine fibrinous flakes and filaments adherent to the surface of the serous membrane or bridging across the space between its two layers. The liquid is sometimes rendered turbid by the presence of desquamated endothelial cells and extravasated leucocytes, and it is occasionally tinged with extravasated blood.

In older and larger hydroceles the liquid is usually clear and colourless or yellow; sometimes it is stained red or brown by admixture with blood, in other cases it is turbid and milky-looking or contains glistening tablets of cholesterin; in others again it becomes inspissated into a white or pigmented pulp containing cholesterin. These latter changes however indicate that the case has passed into one of plastic haemorrhagic periorchitis, and is no longer an example of mere serous periorchitis.

The liquid of the hydrocele not infrequently contains spermatozoa (spermatic hydrocele). This is in some instances due to the rupture of a coexisting spermatocele into the sac of the hydrocele. According to M. ROTH, however, it is oftener brought about by the presence of a vas aberrans from the head of the epididymis, which is situated at the attachment or near the free extremity of the hydatid of Morgagni and opens directly into the vaginal sac. Accordingly in such cases the spermatic hydrocele is a result of some congenital anomaly of development.

In old hydroceles the tunica vaginalis is usually more or less thickened. In many cases its inner surface is beset with *plaques* and ridges of dense fibrous tissue, showing that the condition has become one of plastic periorchitis.

The epididymis generally, and the testis occasionally, become indurated and atrophic; but this is usually due not to the hydrocele itself but to antecedent epididymitis and orchitis, of which the hydrocele is one of the results. The pressure of the hydrocele has remarkably little effect on the parenchyma of the testis.

Serous periorchitis is usually unilateral, and is sometimes observed even in cases where the testis is abnormally situated (inguinal hydrocele). When the vaginal process remains open the contents of the hydrocele may be forced into the abdominal cavity (congenital or peritoneo-vaginal hydrocele). In some instances the sac of a hydrocele is constricted into two compartments connected by an opening or channel, the result being a bilocular hydrocele. Multilocular hydroceles have occasionally been met with.

Serous liquid sometimes collects in the persistent vaginal process accompanying the spermatic cord, owing to serous perispermatitis (KOCHER), and gives rise to **encysted hydrocele of the cord** (Fig. 502 *a*): this may exist either alone or in combination with vaginal hydrocele (infantile hydrocele); it may be shut off on all sides or communicate with the abdominal cavity (congenital hydrocele of the cord); and it may be situated within or without the inguinal canal, and occasionally extends so far downwards as to thrust aside the testis (extravaginal hydrocele).

When liquid accumulates in the sac of an inguinal hernia, and forms a visible swelling, it is called a **hernial hydrocele.**

Purulent periorchitis usually makes its appearance as a result of wounds of the tunica vaginalis or of suppuration of the epididymis and testis; it is very rarely haematogenous in origin. It may supervene either in an already inflamed tunica vaginalis, as in a hydrocele which has been punctured, or in a membrane that was previously healthy. It is characterised by the gathering of pus in the vaginal sac and by the formation of fibrino-purulent deposits on the tunica vaginalis. When septic infection is superadded the effusion is liable to become putrid. Recovery takes place by the production of granulation-tissue and adhesions between the apposed surfaces of the sac.

Purulent inflammation of the persistent vaginal process accompanying the cord is known as purulent perispermatitis.

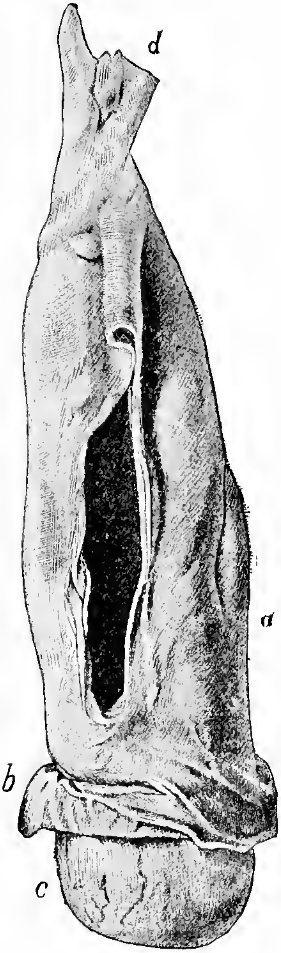

FIG. 502. HYDROCELE OF THE SPERMATIC CORD.

a sac
b epididymis
c testis
d spermatic cord

Plastic periorchitis is as common an affection as hydrocele: it is characterised by the formation of new connective tissue on and between the layers of the tunica vaginalis. It sometimes originates in a previously healthy membrane as a result of traumatic haemorrhage, epididymitis, etc.; in other cases it appears in an already altered membrane, as for instance after a hydrocele has been tapped and the sac washed out. As in the plastic forms of pleurisy or peritonitis, it probably always begins with the deposit of coagulated fibrinous exudations on the serous membrane. Then vascular granulations and fibrous tissue are formed, and penetrating the deposits ultimately take their place. When the process has gone on for a time, both surfaces of the tunica vaginalis are indurated and beset with thick rigid plates and ridge-like prominences of dense fibrous tissue, often impregnated with calcareous salts. KOCHER has applied the term **proliferous periorchitis** to this variety of the affection.

Membranous and cord-like adhesions are frequently formed between the inflamed and thickened layers of the tunica vaginalis, which at times are so abundant as to fill up and obliterate its cavity, a condition fitly described as **adhesive periorchitis.** When owing to fresh inflammatory effusions a large amount of liquid accumulates in the interstices of the adhesions, a peculiar variety of multilocular hydrocele is produced.

The blood-vessels of the newly-formed connective tissue and adhesive membranes are at first wide and thin-walled, and accordingly slight disturbances of the circulation, minor injuries, and the like, are liable to give rise to haemorrhage: this sometimes takes the form of small ecchymoses, sometimes of copious effusions into the sac, and results in red or brownish coloration of its liquid contents, the formation of fibrinous clots and laminated membranes, and pigmentation of the tissues. The inflammation is apt to be rekindled by the presence of the extravasated and coagulated blood, and the production of new tissue is thereby started afresh.

It thus happens that plastic periorchitis combined with recurrent haemorrhage (haemorrhagic periorchitis) is the form which leads to the most extensive changes in the affected parts. The thickening of the tunica vaginalis is extreme, the fibrous plates upon it are often calcified or even ossified, and the tensely-distended sac forms a bulky tumour. The inner surface of the sac is covered with layers of stratified fibrin, here and there interpenetrated by ingrowths of granulation-tissue. The liquid occupying the sac and infiltrating the fibrinous masses is clear and colourless, or tinged yellow, red, or brown; in rare cases it is rendered milky by admixture with fat-globules. A sac filled with blood-stained liquid is termed a **haematocele.** The contents often include cholesterin-plates, or take the form of a pulpy mass of fatty detritus and cholesterin, whose colour is either white, or brownish from the presence in it of granular blood-pigment.

The epididymis is usually much indurated and often so embedded in the thickened wall of the sac that it can scarcely be recognised. The testis still projects into the lumen of the sac, but it is so swathed in thick layers of fibrous tissue that at last it looks like a mere lenticular thickening on the wall, and its glandular tubules are more or less atrophied.

Chronic plastic haemorrhagic perispermatitis is a similar process affecting the persistent remnant of the vaginal process in the spermatic cord within or without the abdomen: it is also described as encysted extravaginal haematocele of the cord.

A peculiar form of plastic periorchitis, characterised by the formation of irregular pedunculated or sessile excrescences and warty overgrowths is known as villous or verrucose periorchitis or vaginalitis (LANCEREAUX). The growths are usually situated on the epididymal portion of the tunica vaginalis, where certain minute villous fringes are almost invariably present in normal conditions (LUSCHKA). When the morbid overgrowths project above the surface, their peduncular attachment to the serous surface sometimes gives way, and they thus form loose bodies within the sac.

Tuberculous periorchitis is usually consecutive to tuberculous disease of the epididymis and testis; but it occasionally appears as an independent affection, in the form both of a disseminated eruption of tubercles and of large granulomatous vegetations resembling those seen in tuberculous bursitis (Art. 87). The eruption of tubercles is liable to be associated with liquid exudation into the vaginal sac.

Syphilitic periorchitis generally accompanies the specific affection of the testis: it leads to fibrous thickening and adhesions of the two layers of the membrane, and may be associated with a form of hydrocele. Gummata of the tunica vaginalis are very rare.

Haemorrhage into the vaginal sac, forming haematoma of the tunica vaginalis, results from contusions and other traumatic injuries, and takes place in connexion with haemophilia, but not as a rule from a previously healthy serous membrane. The commonest cause is indeed injury to a pre-existing hydrocele, which is thereby converted into a haematocele. The blood thus effused into the tunica vaginalis, and the coagula it forms, may remain for a long time unchanged; but sometimes the blood becomes decolorised and its liquid portions are re-absorbed. The remaining coagula set up inflammation, and this results in the formation of new fibrous tissue on the serous surfaces.

Dropsy of the vaginal sac is not infrequent in connexion with general anasarca. Lymphorrhagia with effusion of a milky liquid into the sac (**galactocele**) is met with chiefly in tropical countries and under the same conditions as lymphorrhagic elephantiasis.

Primary tumours of the visceral tunica vaginalis are rare. Cases of fibroma, sarcoma, myxoma, rhabdomyoma, and dermoid cysts have been observed. Of the animal parasites, *Echinococcus* has more than once been met with in this membrane.

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GENZMER: Hydrocele *Volkmann's klin. Vorträge* no. 135 Leipzig **1878**
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HUMPHRY and JACOBSON: *Holmes' System of surgery* III London **1883** (with references)

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Poisson: Periorchitic fibromata *Thèse* Paris **1858**
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CHAPTER CIII

THE SEMINAL TRACT AND THE PENIS

311. The **spermatic cord** (*funiculus spermaticus*) is a structure extending from the testis to the internal abdominal ring. It is enclosed by the funicular process of the tunica vaginalis communis, a fibrous sheath derived from the transversalis fascia and carried down by the testis as it descends. The cord encloses the vas deferens, and the arteries, veins, lymphatics, and nerves going to and coming from the epididymis and testis.

The **vas deferens** or excretory duct of the testis is lined with columnar epithelium and surrounded by a stout layer of muscular tissue. As it enters the fundus of the seminal vesicle it widens into the so-called ampulla, which sometimes gives off a few blind diverticula, and is provided with special tubular glands.

The **seminal vesicles** are appendages to the vasa deferentia, and consist of irregularly-sacculated receptacles whose mucous membrane contains glands, and which branch off from the ducts near their entrance into the prostate. Between its junction with the seminal vesicle and the prostate the vas deferens is straight, and this portion is described as the ejaculatory duct.

The most important lesion of the vas deferens is **inflammation** (spermatitis) appearing in connexion with inflammation of the urethra, prostate, bladder, and epididymis. It usually takes the form of mucous and purulent catarrh (as in gonorrhoea). Occlusion of the duct occasionally results from ulceration or traumatic injury.

Tuberculosis of the urogenital system may involve various parts of the vas deferens, such as that contained in the spermatic cord, that within the pelvis, the ampulla, or the ejaculatory duct. The mucous membrane, the muscular coat, and finally the fibrous adventitia are successively attacked, and undergo caseous degeneration, necrosis, and ulceration.

Gummata of the duct have been observed in a few cases (VERNEUIL, KOCHER).

The seminal vesicles are usually affected in sympathy with the vas deferens. In catarrhal inflammation their interior becomes filled with mucus or muco-pus, and in tuberculous disease with caseous detritus. The walls are more or less thickened by cellular or granulomatous infiltration as the case may be, and in advanced tuberculosis they are generally caseous.

The contents (whether normal or inflammatory) of the seminal vesicles, when their evacuation is obstructed, are liable to become inspissated and calcareous, and in this way concretions and calculi are produced. Spermatozoa have more than once been discovered in these concretions (spermatic calculi).

The **veins** of the spermatic cord not infrequently become varicose throughout, their minute ramifications in the testis, epididymis, and lining mucous membrane being alike dilated. In this manner the apparent bulk of the spermatic cord becomes enlarged, and a **varicocele** is formed. It is often due to the impeded efflux of blood from the cord occasioned by the pressure of a tumour, hernia, or the like.

Swelling of the spermatic cord arising from interstitial oedema of its tissues has been called **diffuse hydrocele** of the cord.

Rupture of the spermatic arteries or veins gives rise to the formation of a **haematoma** of the cord, the effused blood occupying the loose areolar tissue between its several constituent structures. The condition usually results from the laceration of varicose veins or rarely of arteries from such causes as traumatic injury, severe coughing, abdominal straining, etc. Haematoma sometimes produces an enormous tumour-like swelling of the cord. The blood is usually diffused throughout the entire structure (diffuse haematoma), and may even spread through the inguinal canal to the subperitoneal tissue of the abdomen; but when the haemorrhage is not copious it gives rise only to a local and circumscribed swelling (circumscribed haematoma). Large haematomata do not in general disappear spontaneously, though they tend in the course of time to become circumscribed. Small extravasations are usually re-absorbed, though they leave some induration and pigmentation behind them.

Inflammation of the vas deferens, and especially that due to tuberculosis, is apt to extend to the other structures of the cord.

Primary tumours of the spermatic cord are rare. Lipoma, fibroma, myxofibroma, and sarcoma have been described. Sarcoma and carcinoma of the testis occasionally produce metastases in the cord.

References on the Morbid Anatomy of the Spermatic Cord and Seminal Vesicles (see also Art. 306).

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HENRY, MITCHELL: Abscess of the seminal vesicles *Med.-chir. Trans.* XXXIII London **1850**
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ZEISSL: Inflammations of the vas deferens *Allgem. Wien. med. Zeit.* XV **1870**; Syphilitic disease of the cord *Wien. med. Blätter* VI **1883**

312. The **prostate** is a glandular organ with an abundant stroma consisting largely of muscular tissue, which surrounds the beginning of the urogenital canal or urethra. It is developed by epithelial outgrowths from the posterior wall of the primitive urogenital sinus, which are transformed into a compact mass of branching tubular and saccular glands lined with columnar epithelium. These are embedded in a muscular stroma overlaid by a thick capsular stratum of unstriped muscle and fibrous tissue.

In children this gland is small, and it does not attain any considerable size until the age of fifteen to twenty-five years. The greater part of the organ is posterior to the urethral canal, and is there divided into two lateral lobes separated by a slight depression. Sometimes there is a small third or median lobe between the two lateral lobes. The portion of the prostate anterior to the urethra is usually small, being sometimes reduced to a mere shallow bridge of fibrous tissue.

The glands from the lateral lobes open along the sides of the longitudinal median ridge rising from the floor of the urethra known as the *colliculus seminalis* or *verumontanum*, the anterior end of which contains the orifices of the ejaculatory ducts. In this situation also there is a median blind recess running backward into the gland for five to ten millimetres, described as the prostatic utricle (*uterus masculinus*) and representing a persistent remnant of the ducts of Müller. This utricle is sometimes abnormally large. According to ENGLISCH, other remnants of Müller's ducts are occasionally dilated into **cysts** lying within the substance of the prostate or close to it.

Total **absence** of the prostate is observed only in cases of extreme malformation of the urogenital system. Its size as a whole, and the proportions of its component parts, are subject to very considerable variations.

Atrophy of the prostate is met with at all ages, and may affect either the stroma or the glandular tissue or both.

Inflammation of the prostate (prostatitis) is often associated with inflammation of other parts of the urogenital system, such as gonorrhoeal urethritis and purulent or putrid cystitis, and with inflammations of the rectum and cellular tissue of the pelvis. Apart from these the commonest exciting cause is traumatic injuries, by instruments for example, haematogenous prostatitis being rare. The inflammatory exudation and infiltration give rise to more or less marked swelling of the organ. In catarrhal inflammation of the glands pressure forces out an opaque whitish secretion from their orifices.

The inflammation usually subsides and the exudations are re-absorbed; induration of the stroma with atrophy of the glands is a rare termination. In purulent inflammation yellowish-white infiltrations are formed, which presently break down into **abscesses.** Small abscesses generally become cicatrised, but larger ones are apt to rupture into the urethra or rectum, or occasionally into the surrounding connective tissue. When they are once evacuated in this way, granulation and cicatrisation may follow.

Prostatic **tuberculosis** is usually consecutive to the like disease in neighbouring parts of the urogenital system, but it is sometimes a primary affection. It may take the form of small grey tubercles or large caseous masses, as the case may be; softened necrotic foci sometimes break into the neighbouring tissues.

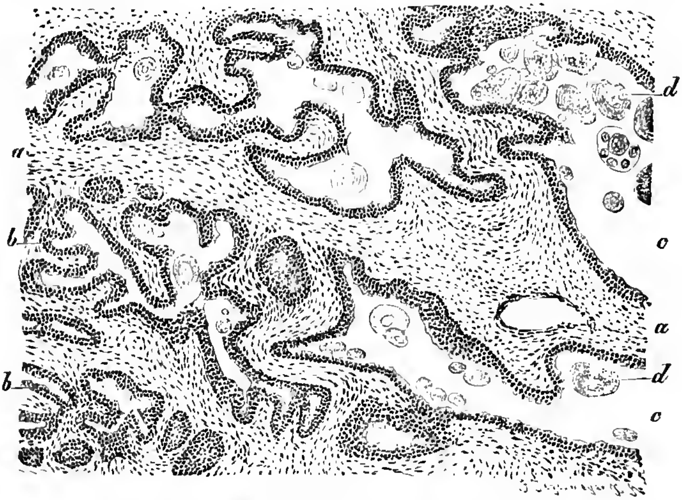

FIG. 503. SECTION OF A HYPERTROPHIED PROSTATE WITH CONCRETIONS.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin* : × 45)

a stroma *b* glands *c* dilated glands *d* concretions

Glanders occasionally gives rise to purulent prostatitis.

In old age the glandular ducts and acini of the prostate nearly always contain **concretions,** the smallest being recognisable only with the aid of the microscope (Fig. 503 *d*), while the larger ones appear as brownish or blackish grains rarely exceeding a milletseed in size, but at times in considerable numbers. Certain of these bodies give with iodine a reaction similar to that characteristic of amyloid degeneration. They sometimes undergo calcification, especially when they are of any appreciable size. They are the result of a peculiar hyaline transformation of the protoplasm of dead and desquamated cells.

The smaller concretions, which resemble *corpora amylacea*, appear in the prostate even in childhood. They are formed from the cells occupying the interior of the solid epithelial outgrowths that represent the rudiment of the gland. These cells break down and are converted into hyaline flakes, which become packed together and by fresh accretions are converted into stratified bodies. The pigment contained in many of them is probably of the same nature as the yellow granules which in older persons are usually found here and there enclosed within the glandular epithelial cells.

According to STILLING, hyaline degeneration of the muscular fibres of the prostate may be observed both in old and in young patients, particularly after febrile affections. In aged persons the outer fibrous layer in the walls of the ducts undergoes a hyaline thickening which sometimes obstructs the channel, and so favours the retention of secretion and the formation of concretions.

Enlargement of the prostate is a very common accompaniment of old age, the whole gland or only particular parts of it being increased in bulk. The cut surface may retain its uniform texture, or exhibit circumscribed patches of a different structure, in which latter case the exterior of the gland is in general irregularly tuberous. When the lateral lobes are the chief seats of enlargement (Fig. 490 *c*) the urethra is apt to be laterally compressed: when one only of the lateral lobes is overgrown the channel is pushed over to the other side; and when the posterior median portion (*b*) is prominent the posterior wall of the neck of the bladder and the first portion of the urethra are driven inwards and forwards. These conditions are in varying degree liable to cause obstruction to the evacuation of the bladder, with consecutive cystitis.

Both in the diffuse and in the nodose form the overgrowth is occasionally confined to the fibro-muscular tissue (fibromyoma), the glandular structures being unaffected, or undergoing simply atrophy and cystic degeneration. In most cases however the glandular tissue also becomes hypertrophic (Fig. 503 *b c*), sometimes to such an extent that it comes to be more abundant than the fibro-muscular stroma, and the process then assumes the character of a glandular hyperplasia. Many authorities indeed describe the enlargement as an adenoma. There is much clinical evidence to show that the glandular hypertrophy is related to the functional activity of the testes, and that castration is often followed by dwindling of the enlarged prostate (ROCUM, WHITE). GRIFFITHS found abundant signs of fatty degeneration of the tubular epithelium with some cellular infiltration of the stroma of the gland in a man who had undergone bilateral castration eighteen days previously.

Destructive **adenoma** and **carcinoma** are somewhat rare; but they occasionally appear in young and old persons, generally as soft nodose growths which project into the lumen of the urethra or of the neck of the bladder, and as they grow invade the contiguous tissues. As the neoplastic tissue breaks down the prostate becomes ulcerous, and copious haemorrhage is a not uncommon result.

Large **valve-like folds** of mucous membrane due to congenital malformation sometimes arise from the colliculus seminalis in the prostatic urethra, and impede the passage of the urine.

Cowper's glands are two compound racemose glands of from six to eight millimetres in diameter, and situated one on either side of the membranous urethra, immediately behind the bulb of the corpus spongiosum. These structures often become inflamed in connexion with urethritis, in which case they swell up and project into the urinary passage: sometimes they actually suppurate. In chronic inflammation they are liable to become permanently enlarged and indurated. When their excretory ducts are obstructed they become dilated into small cysts of retention, which in children are capable of interfering with free micturition (Elbogen).

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313. Apart from its cutaneous covering the **penis** consists of the common urogenital canal or *urethra*, and of the *corpora cavernosa*, the latter being developed from the genital tubercle in front of the foetal cloaca. Around the urethra the muscular wall of the urogenital canal is converted into a spongy mesh-work of wide anastomosing vascular sinuses, the *corpus spongiosum*, through the upper part of which the urethra runs, its tube being bounded by a mucous membrane and surrounded by a thin layer of muscular tissue continuous with the muscular walls of the sinuses. At the proximal end the corpus spongiosum expands into the bilobate *bulb;* at the distal end it is rounded into the *glans penis.*

The corpora cavernosa of the penis, which are invested with a stout fibrous sheath, are attached by their divergent *crura* to the rami of the os pubis and ischium. They lie along the dorsal or superior surface of the urethra, above which they are closely united, and end in the coronary sulcus behind the glans penis. Their blood-vessels are larger and more irregular in form than those of the corpus spongiosum. At the anterior extremity of the organ the outer integument is infolded so as to invest the glans with a movable covering known as the **prepuce** or foreskin.

Complete **absence** of the penis is very rare, and occurs only in association with other grave defects of development in the external genitals. Duplication of the penis, or of the urethral canal in a single penis, one channel being urinary and the other seminal, is also rare. Extreme dwarfing of the penis, so that it somewhat resembles the clitoris, is more common, and is generally associated with hypospadias or backward misplacement of the urethral orifice (Fig. 453).

Hyperplasia of the prepuce is not uncommon; if it is associated with contraction of the external orifice, so that the fold of skin cannot be retracted over the glans, the condition is described as hypertrophic phimosis. Complete absence of the prepuce is rare; but abnormal shortness of this fold is more frequently met with.

Herpes, eczema, soft chancre or chancroid, syphilitic or hard chancre, mucous patches or papules (*condyloma latum*), erysipelas, tuberculosis, elephantiasis, venereal warts (*condyloma acuminatum*), cutaneous horns, and carcinoma are met with on the prepuce and glans penis. They are dealt with in connexion with diseases of the skin (Section VIII).

Tuberculosis of the glans penis sometimes gives rise to extensive ulcerative destruction of its tissue.

Carcinoma appears most frequently in the glans and prepuce, and takes the form of exuberant papillary outgrowths, or of an indolent ulcer. Sometimes the entire penis is invaded and destroyed by the cancerous infiltration.

Inflammation of the glans penis is known as **balanitis**, that of the inner fold of the prepuce as **posthitis.** They may be induced by one of the cutaneous inflammations above mentioned, but are not infrequently set up by the accumulation beneath the prepuce of decomposing smegma or urine, of infective pus from the kidney or bladder, or of purulent urethral discharges in cases of gonorrhoea or chancre. In diabetes the growth of *Aspergillus* beneath the prepuce sometimes excites inflammation there. Posthitis is generally accompanied by considerably inflammatory oedema of the prepuce, which cannot be retracted from the glans. This condition is known as inflammatory **phimosis**; and when on the other hand the foreskin, once retracted, becomes so oedematous that it cannot be pushed forward over the glans, the result is **paraphimosis.** Exceptionally severe inflammation is apt to end in ulceration or even in gangrene of the glans and prepuce. Adhesions between the two are occasionally produced during the healing of the ulcerations.

Calculi are sometimes formed under a narrow prepuce from inspissated smegma and desquamated epithelium, by incrustation of these with urates, calcareous salts, etc. Urinary calculi may also be retained beneath the prepuce, and become enlarged by subsequent accretion *in situ.*

Lacerated and contused wounds are the most important lesions of the corpora cavernosa, inasmuch as they lead to copious haemorrhage, and on healing give rise to deformity of the penis from cicatricial induration and contraction of the cavernous tissue.

Inflammation of the corpora cavernosa generally results from traumatic injury or from cutaneous or urethral inflammation: it may also however appear in the course of various infective diseases, such as pyaemia, small-pox, typhoid fever, etc. Suppuration and gangrene occasionally supervene, and when repair takes place the penis is often distorted by cicatricial contraction. Nodose thickenings are now and then left behind when the inflammation subsides, and in rare cases partial ossification has been observed in the fibrous trabeculae, and particularly in the median *septum pectiniforme.*

The **scrotum** is a sac composed of two pouches of skin united in the median line by the *raphe.* Beneath the skin lies the contractile dartos tunic, consisting of a layer of smooth muscular fibres. The morbid conditions of the scrotum correspond to those of the skin generally. Elephantiasis, eczema marginatum, and carcinoma (particularly in chimney-sweeps and paraffin-workers) are the most frequent local affections. Elephantiasis or **lymph-scrotum**, due to *Filaria*, is common in Eastern Asia and Africa. The scrotum, by lymphangiomatous hyperplasia of its skin and subcutaneous tissue, becomes enormously enlarged, the tumour sometimes measuring as much as one metre in diameter.

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Kraske: Tuberculosis of the penis *Ziegler's Beiträge* x **1891**
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Niehus: Chronic induration of the cavernous tissue (epithelioma) *V. A.* 118 **1889**
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CHAPTER CIV

THE OVARIES

314. Shortly after puberty the **ovary** appears as a somewhat flattened oval organ of from three to five centimetres in length; it lies within the true pelvis embraced by a fold of peritoneum proceeding from the posterior layer of the broad ligament. The greater part of the organ stands out from the posterior surface of the broad ligament into the abdominal cavity, and is uncovered by the peritoneum, which ends abruptly by a definite line of demarcation (the white line of FARRE) at the base of attachment or *hilum* of the ovary.

Absence of both ovaries is met with only in association with other grave malformations of the genital system. Absence of one ovary is in like manner usually combined with some anomaly of the tube and uterus on the same side (as in *uterus bicornis* or *unicornis*), though cases have been recorded in which both uterus and tubes were normal. In certain cases the absence of the organ is attributable to constriction from torsion of its pedicle or from morbid adhesions.

Hypoplasia of the ovaries is by no means rare, appearing both during the period of foetal development and during adolescence. The size and form of the ovary vary much even within physiological limits; the proportion of length to thickness is the most variable, but the bulk of the organ as a whole is also subject to considerable fluctuation. The ovaries may fairly be described as hypoplastic when during the years of sexual maturity they are no larger than in childhood, or when their follicles altogether fail or are much retarded in ripening. The primary follicles and ovarian ova are in such cases usually scanty, the organ consisting mainly of highly-cellular connective tissue with few or no visible vesicles.

The hypoplastic condition is commonest in cases of general dwarfing and undergrowth of the whole body, in cretinism, and in chlorosis (VIRCHOW). The other genital organs in these cases may be either fully developed or undergrown. The development of the genital passages does not appear to depend on the complete evolution of the ovarian structures.

Hypertrophy of the ovaries is deemed to exist when the organs exceed in size the dimensions given above as maximal, and at the same time contain an excessive number of follicles (Art. 315). Cases have been recorded in which the ovaries measured as much as six to eight centimetres in length. In a certain sense the precocious ripening of the follicles in childhood, a not uncommon occurrence even in new-born infants (SLAVJANSKY, DE SINÉTY) might be regarded as evidence of hypertrophy.

Supernumerary ovaries have been observed in many cases (GROHE, KLEBS, DE SINÉTY, WINKEL, OLSHAUSEN), the multiplicity being due either to spontaneous subdivision of the embryonic rudiment of the organ, or to mechanical abstriction of one or more portions from it. Occasionally the surface of an ovary is raised into one or more small button-like prominences which are sometimes separated from the rest of the stroma by deep grooves lined with columnar epithelium.

During its normal descent into the pelvis an ovary now and then passes with the vaginal process of the peritoneum into the inguinal canal, and may even escape through the external ring into the labium majus. In later life it is sometimes dragged into the sac of an inguinal hernia, and may reach the exterior of the pelvis through the femoral canal, the sacro-sciatic notch, or the foramen of the obturator membrane (KIWISCH). The posterior wall of the vagina sometimes prolapses, and the ovary occupying the pouch so formed may pass into the vagina or even protrude through the vulvar orifice (vaginal ovariocele); but all such extreme displacements are rare.

The free surface of the ovary is covered with a single stratum of columnar epithelium (the germinal epithelium of WALDEYER), under which lies a firm layer of fibrous tissue, the *tunica albuginea*. Beneath this membrane we find a much broader zone of tissue known as the cortical or follicular layer, the parenchyma of the organ (WALDEYER), which is interrupted only at the hilum. Through the hilum the blood-vessels, lymphatics, and nerves enter from the broad ligament. The fibro-cellular stroma of this layer is studded with *follicles*, whose diameters vary from 0·042 to 15·0 millimetres. The smaller or primary follicles, varying from 0·042 to 0·15 millimetres in diameter, lie principally in the outer portions of the parenchymatous zone, and consist of a single layer of low columnar epithelial cells enclosing an ovum, which occupies the centre and completely fills the follicle. The follicles of the next larger size are lined with several layers of cubical epithelial cells at the part in contact with the ovum. Still larger follicles, of from 0·5 to 5·0 millimetres in diameter, are completely invested on their inner surface with a stratified lining of cubical epithelial cells (*membrana granulosa*), enclosing a central cavity filled with liquid (*liquor folliculi*). In these the ovum is situated eccentrically, and is supported by a cellular cluster which in the case of the largest follicles projects into the cavity (*cumulus* or *discus proligerus*), the cells surrounding the ovum forming a single columnar layer that is firmly adherent to it. The ovum in the full-sized follicles is of appreciable volume (0·2 mm. in diameter), and is enveloped in a radially-striated membrane (*zona pellucida* or *striata*).

The follicles that possess a central cavity are known as *graafian follicles*. They are separated from the surrounding stroma by a wall of fibrous tissue (*theca folliculi*), consisting of an outer fibrous layer (*tunica fibrosa*) and an inner vascular and cellular layer (*tunica propria*).

The larger graafian follicles are situated in the deeper strata of the parenchyma of the organ. As they increase in size they approach the surface, pushing aside the smaller immature follicles, and finally protrude from the surface of the organ invested only with a fine layer of fibrous tissue, the most prominent portion of which is very thin and contains no vessels (*stigma* or *macula pellucida folliculi*). An ovary contains only a small number of the larger follicles: at most one or two of the dimensions given above as the maximum are normally present at any one time.

The central portion of the ovary about the hilum, sometimes called the medulla, is very vascular, the vessels indeed predominating over the fibrous elements. The arteries have a spiral or flexuous course, and the veins are wide. The stroma contains non-striated muscle-cells that proceed from the suspensory ligament.

The development of the specific constituents of the ovary begins in the third month of foetal life by the ingrowth, in the form of processes or blind tubes, of the ovarian germinal epithelium into the contiguous connective tissue of the future cortical layer. These 'egg-tubes' (PFLÜGER) ramify and intercommunicate, forming a network of cellular strands, which are broken up into small cellular nests or clusters by their interpenetration with the proliferous fibrous stroma, and are thus converted into primary follicles, each containing a large central cell. The ingrowth of the germinal epithelium continues for a time after birth. According to PALADINO, the ovarian parenchyma is continually being reproduced in this way up to the establishment of the menopause. According to other authorities (KOSTER, GUSSEROW, NAGEL) the abstriction by the stroma of portions of the germinal epithelium that takes place in later life is to be regarded as pathological, and related to chronic inflammatory processes.

The ovary in the new-born infant is a comparatively long flattened organ with a surface that is usually smooth, but is not infrequently pitted and uneven. As the infant grows older the organ increases in size, and reaches its fullest dimensions after puberty. Small scattered vesicles containing liquid sometimes appear even in the infantile ovary, but completely-mature follicles are not produced until puberty is established.

The number of primary follicles in the ovary is greatest in infancy, at the period when their development is just completed. From that time onwards the number decreases. The majority of the ova perish and are absorbed within the ovary, and most of the follicles also disappear without discharging their contents. The process of retrogression is accomplished by the resorption of the follicular contents, which are displaced by large cells derived by proliferation from the follicular wall. In the larger follicles the contents are penetrated by stellate fibroblasts and so converted into fibrous tissue. The large-celled tissue derived from the theca folliculi is also in the end transformed into fibrous tissue resembling that of the ovarian stroma.

Some of the follicles, attaining maturity and so growing into large vesicles, evacuate their contents into the abdominal cavity, and the ova thus extruded are in normal conditions taken up by the fallopian tubes. The extrusion usually takes place at the time of menstruation, rarely at other times, and is brought about by an increase in the contents of the vesicle, which distends and attenuates the overlying parts of the theca folliculi and tunica albuginea at the surface of the ovary, and ultimately ruptures them. If the rupture is unaccompanied by haemorrhage the follicular cavity becomes filled with a gelatinous mass. But if as is usually the case some haemorrhage does take place, the cavity is chiefly occupied by clotted blood which by and by assumes a brownish tint.

Even before the follicle ruptures, the inner layer of the theca folliculi undergoes a certain amount of hypertrophic proliferation; after rupture the proliferation becomes more active, and the blood-stained or gelatinous contents are presently enclosed in and displaced by a comparatively thick envelope, which is thrown into plaits or folds as it grows, and is composed of large embryonic cells and new-formed blood-vessels. The cavity is reduced by degrees to a mere irregular cleft, the yellowish folds of the envelope cohere and practically fill the follicle, and it is thereby converted into a *corpus luteum.*

When it is fully developed, namely in the second or third week after the rupture of the follicle, the corpus luteum measures from 8 to 15 millimetres in diameter. Its size is largely dependent on the quantity of blood-clot it encloses. When the ovum perishes unimpregnated, the corpus luteum in the course of a few months undergoes retrogressive changes, the proliferous embryonic tissue derived from the theca folliculi, and ultimately the theca itself, being transformed into tissue similar to that of the surrounding stroma. The central mass (Fig. 505 *c*) becomes a homogeneous and lustrous kernel of fibroid non-cellular tissue (*corpus fibrosum*), which also after a time merges into the stroma and disappears, at least in women that are not advanced in years.

When pregnancy supervenes, the retrogression of the corpus luteum does not begin until the middle of gestation, and the process is not completed until some months after delivery.

By the cicatrisation of the follicles as they successively rupture, the surface of the ovary becomes more and more uneven, being dinted and furrowed with small scars.

After the menopause the ovary diminishes considerably in bulk, and becomes flatter in the sagittal direction. The germinal epithelium at its surface persists, but the more or less mature ova and follicles usually perish. Sometimes however a few isolated follicles may be found even in advanced age. Corpora fibrosa are probably never absent in the ovaries of old women, and are often very numerous. This is no doubt due to the fact that with the cessation of ovulation the retrogression of the corpora lutea is no longer completely accomplished. The vessels of the stroma at the hilum sometimes undergo considerable thickening of their intima, or even hyaline degeneration and calcification of the entire wall.

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315. Most of the morbid changes in the ovaries take place during the period of their greatest functional activity, and some of them are directly related to the processes of maturation and ovulation. The congestive hyperaemia of the genital organs associated with menstruation and coition occasionally leads to **haemorrhage** into the ovarian follicles and even into the stroma. Slight extravasations give a reddish tinge to the contents of the follicles, which appear as small haemorrhagic spots in the stroma. Larger haemorrhages may distend the follicles to the size of a walnut; and when the parenchyma also is infiltrated the entire organ is apt to be considerably swollen. Copious haemorrhage into a ruptured follicle sometimes leads to the effusion of blood into the abdominal cavity, where it collects in Douglas's pouch and there coagulates (pelvic haematocele). If adhesions have previously been formed about the ovaries and uterus, the effused blood collects in the spaces between the adhesive membranes and bands (retro-uterine haematocele). A blood-clot lodged in the pelvic peritoneal pouch sets up inflammation and proliferation about it, and fresh adhesions are thus formed between the contiguous organs and structures.

Free haemorrhage into the follicles kills the ova they contain, while copious extravasation into the stroma destroys the smaller unripe follicles and it may be the tissue of the stroma itself. Cystic degeneration and obliteration of the follicle seem occasionally to follow haemorrhage into it, and atrophic patches are left behind in the parts of the stroma where primary follicles have thus been destroyed.

Ovarian haemorrhage is moreover apt to occur in cases of inherited or acquired haemophilia or scurvy, in various infective fevers, and in cholera.

In **leukaemia** the ovarian blood-vessels become filled with colourless blood-corpuscles: these are occasionally extravasated into the parenchyma, and appear in the form of streaks following the course of the vessels or of nodular aggregations of cells.

When a large number of follicles reach maturity at the same time, or when the follicles reaching maturity in normal succession fail to rupture, the ovary at length looks as if it were made up entirely of small cysts, and becomes greatly enlarged. This appearance has often been described as cystic degeneration, but so long as the mature follicles do not exceed the normal size and still contain ova the term is inapplicable, the condition being really one of **follicular hypertrophy.** Why the follicles should fail to rupture cannot as a rule be made out with certainty. In many cases some abnormal toughness of the follicular membrane and thickness of the tunica albuginea seem to be the cause.

Inflammation of the ovary, or **oöphoritis,** is usually due to extension of some inflammatory process from the uterus, tubes, and peritoneum (Fig. 504), or occasionally from other adjacent structures; in some instances however it appears to follow upon haemorrhage into the parenchyma of the ovary itself. Very few observations have been made concerning haematogenous inflammations of the ovary, but in connexion with the acute exanthemata, typhoid, septicaemia, cholera, and poisoning by phosphorus and arsenic, certain degenerative processes characterised by cloudy swelling and fatty degeneration of the follicular epithelium and ova, and certain inflammatory changes such as acute oedema, make their appearance, with the result that the contents of the follicles become turbid and the ova are destroyed.

In severe acute inflammation, such as accompanies uterine pyaemia and septicaemia after parturition, the ovary is liable to become greatly swollen and soft or doughy in consistence. Its tissue is more or less reddened and sodden (serous oöphoritis), and at times beset with haemorrhagic patches (haemorrhagic oöphoritis). The follicles often contain a turbid puriform liquid. When actual suppuration sets in (purulent oöphoritis) the ovary is speckled with ill-defined yellow spots and streaks within which the tissue is broken down and liquefied, and ragged **abscesses** are thus produced. Occasionally the entire organ undergoes suppurative necrosis (necrotic oöphoritis). In other rare instances the inflammatory process is practically confined to particular follicles, whose contents become turbid as pus collects within them.

If the suppuration is not fatal, the ovarian abscess tends to become encapsuled by the formation of granulations and new connective tissue around it. The abscess-wall often continues to secrete pus, and the sac is thus distended to a considerable size.

Ovarian abscesses not due to puerperal septicaemic infection are somewhat rare. They are usually secondary to purulent (gonorrhoeal) inflammation of the uterine or tubal mucous membrane, to pyaemic suppuration after operations on the pelvic organs, or to purulent peritonitis starting in some other part.

Chronic oöphoritis, indicated by persistent inflammatory

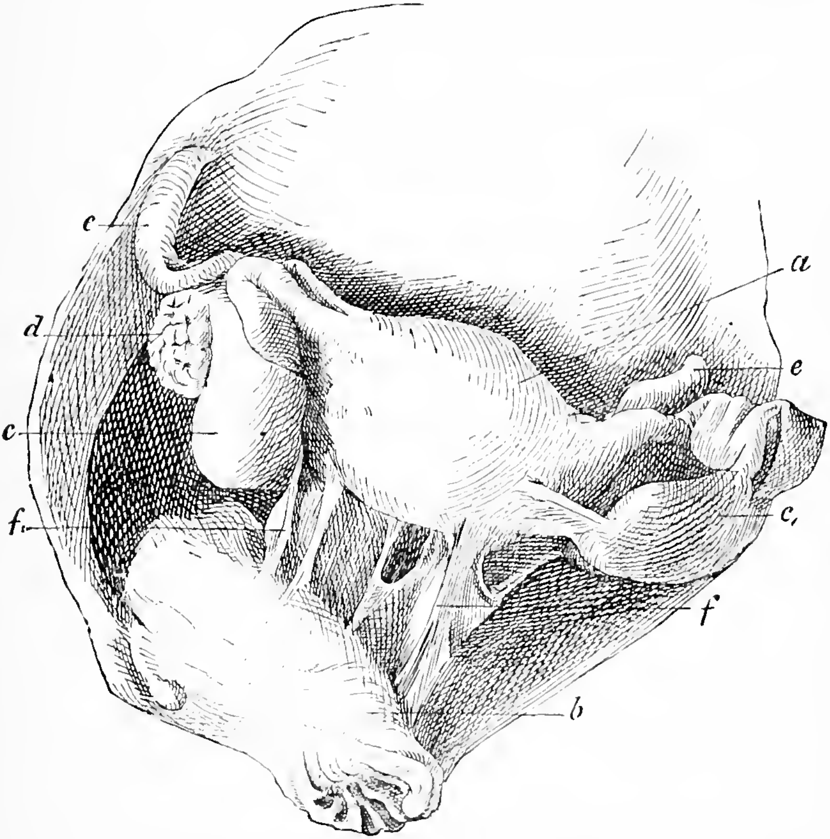

FIG. 504. PERIMETRITIC ADHESIONS OF THE UTERUS, HYDROSALPYNX, AND ATROPHIC CONTRACTION OF THE OVARY.

(From a woman aged 43: five-sixths of the natural size)

a	uterus
b	rectum
c c_1	dilated tubes
d	ovary
e	thickened round ligament
f	membranous adhesions between uterus and rectum

infiltration of the ovarian stroma, is not at all common; but permanent textural changes in the ovary frequently follow acute puerperal and non-puerperal inflammations, and it is to these that the term chronic oöphoritis is generally applied.

The menstrual congestive haemorrhage into the ovary above referred to leads to a more or less wide-spread obliteration of the follicles, and to induration of the stroma, these changes being naturally more pronounced when the ovary is already the seat of diffuse inflammatory infiltration. This variety of inflammation likewise is generally associated with the puerperal state; but acute and chronic inflammations of the uterus, tubes, pelvic peritoneum, rectum, caecum, and vermiform appendage, sometimes bring about transient oöphoritis resulting in permanent textural change.

Traces of previous inflammation of the ovary are often visible even on the exterior. The organ (Fig. 504 *d*) is attached to the surrounding parts, particularly to the tubes and uterus, by loose stringy or ribbon-like bands, or by short and tense adhesions, whereby it is often dragged from its place and brought close to the uterus or even to the bottom of Douglas's pouch. Sometimes it is so entirely embedded in membranous adhesions as to be no longer traceable without dissection.

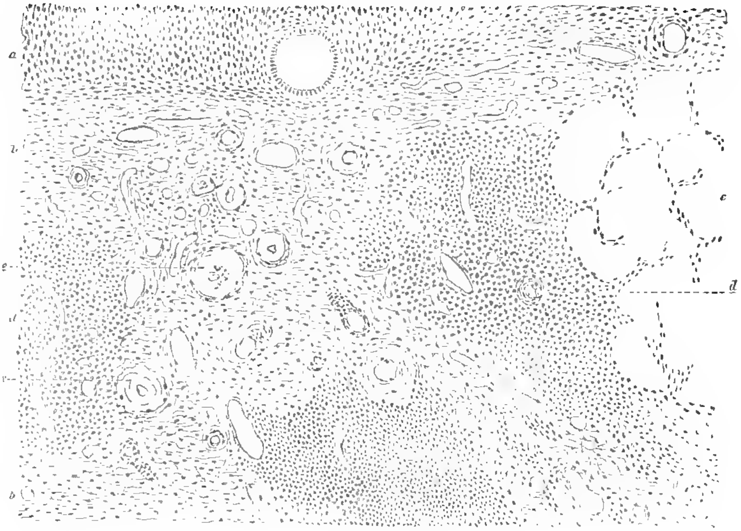

FIG. 505. SECTION OF A CONTRACTED AND ATROPHIC OVARY WITH PATCHES OF CELLULAR INFILTRATION.

(*Preparation made from the ovary shown in Fig.* 504; *hardened in Müller's fluid, stained with alum-carmine and neutral carmine, and mounted in Canada-balsam:* × 40)

a	atrophic cortical substance with a single follicle	*d*	hyperaemic patches of cellular infiltration
b	medullary stratum	*e*	blood-vessels whose walls have undergone hyaline degeneration
c	corpus fibrosum		

Such changes are no doubt mainly due to **perioöphoritis,** but inflammatory changes in the ovary itself are not infrequently associated with the latter, and these can be attributed only to simultaneous or consecutive oöphoritis.

The chronically inflamed ovary is corrugated by numerous depressed cicatrices (Fig. 504 *d*), and is usually gnarled and shrunken: sometimes it is reduced to a shrivelled and distorted knob no larger than a cherry. The changes in its parenchyma are essentially atrophic in character, in other words its follicles are prematurely withered and effaced (Fig. 505 *a*); but some enlarged and cystic follicles with thickened walls are generally present also. Sometimes the tunica albuginea is likewise thickened by the superposition on it of dense fibrous tissue. Textural changes in the stroma of cortex or medulla, that might be taken as indications of antecedent inflammation in it, are not very often met with; but now and then cases occur in which the atrophic tissue is pervaded by aggregations of small round-cells (Fig. 505 *d*) lying chiefly in the zone about the hilum. True cicatricial tissue is seldom apparent, if we except the corpora fibrosa (*c*); this is doubtless due to the fact that the ovarian stroma has a notable power of reproducing itself, and so of effacing the traces of past inflammation. On the other hand, in such shrunken ovaries some of the vessels show signs of hyaline degeneration and thickening in their walls (*e*), and others are actually occluded or obliterated.

Tuberculosis of the ovary is very rare, but it is sometimes observed either as an independent affection or associated with tuberculous disease of the uterus or tubes. The organ is studded with small nodules and larger caseous patches whose centres are softened and disintegrated, and is sometimes enlarged to the size of a hen's egg (GUSSEROW).

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316. A mature follicle which fails to rupture, and does not pass through the usual retrogressive changes, is apt to become enlarged by what is known as **follicular dropsy** (Fig. 506 *d*).

The conditions under which the dropsical enlargement takes place are not fully understood; probably the theca folliculi or the tunica albuginea is so abnormally resistent that rupture is prevented. The contents of the enlarged follicle are usually clear, and resemble the normal liquor folliculi, but occasionally they are tinged red or brown by admixture with blood or the products of its disintegration.

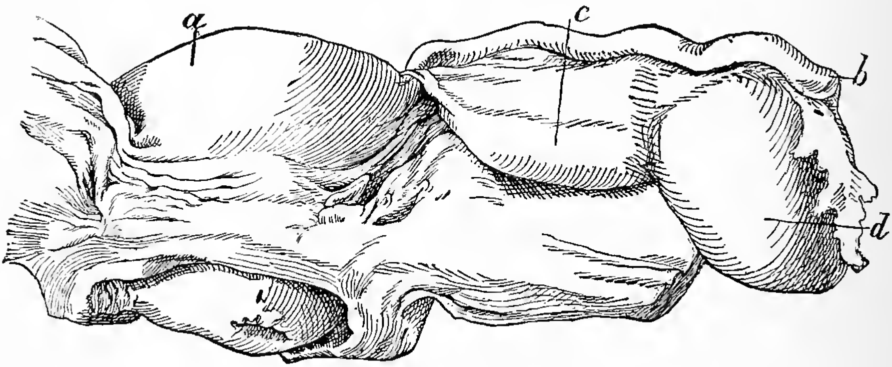

FIG. 506. FOLLICULAR CYST OF THE OVARY WITH RETROFLEXION OF THE UTERUS.
(*Five-sixths of the natural size*)

a uterus *b* tube *c* ovary *d* follicular cyst

Should many of the follicles thus become dropsical the ovary enlarges more or less uniformly to the size of the fist. Usually however one follicle only (Fig. 506 *d*) is distended, the others remaining unchanged. In the course of time this may grow into a cyst, whose size in certain not very common cases may be as great as that of a man's head. Two or more adjacent follicular cysts sometimes coalesce into one by the atrophy of the intervening septum. When the liquid contents of the follicle increase beyond a certain point the ovum it contains is usually destroyed. The follicular epithelium however persists, and lines the cyst with a single layer of flat or more rarely of tall columnar cells. Outside the epithelial lining lies a capsule of fibrous tissue derived from the theca folliculi. As the cyst increases in size a certain amount of fibrous hyperplasia takes place, so that the wall of a large cyst is thicker than that of a mature follicle. When the hyperplasia does not keep pace with the enlargement of the cyst, the most prominent portion of its wall becomes thinned, and may at length give way.

Extreme cystic enlargement of the follicles, by the mechanical pressure and traction it produces, probably in all cases results in atrophy of the rest of the ovarian parenchyma; though at times even when somewhat large cysts are present a few follicles containing ova can still be discovered in the solid tissue. The larger the primary cyst the more does the surviving ovarian tissue assume the appearance of a mere appendage to or patch upon the cyst-wall.

Cystic degeneration of the ovary may be unilateral or bilateral, and the organ may be free or bound by adhesions to the surrounding parts. As such adhesions occur in connexion even with slight enlargements, it is probable that some follicular cysts at least are due to inflammatory changes in the ovaries and their environing structures. When the fimbriated extremity of the fallopian tube is firmly adherent to the ovary, the rupture of a normal or cystic follicle into it sometimes results in the formation of a **tubo-ovarian cyst**, whose wall is composed partly of the dilated tube and partly of the follicular membrane. The free removal of the liquid within the cyst, probably secreted for the most part by the tubal mucous membrane, is sometimes hindered by occlusion or derangement of the uterine orifice of the tube. Even when the orifice is normal it is so placed that the accumulated secretion cannot escape into the uterus continuously, but only from time to time (profluent dropsy of the ovary).

Follicular dropsy very seldom makes its appearance except during the period of generative activity, and most of the simple cysts of the ovary are merely distended follicles. It is very doubtful whether large cysts ever arise from the corpora lutea, in which however small cystic cavities are now and again met with.

Parovarian cysts are due to distension of the tubules of the parovarium by accumulated secretion. They lie between the layers of the tubo-ovarian (broad) ligament, and occasionally reach a very considerable size. Owing to the fact that some of the parovarian tubules are at times continued into the stroma of the ovarian hilum, cysts arising in these aberrant tubules are difficult to distinguish from true ovarian cysts.

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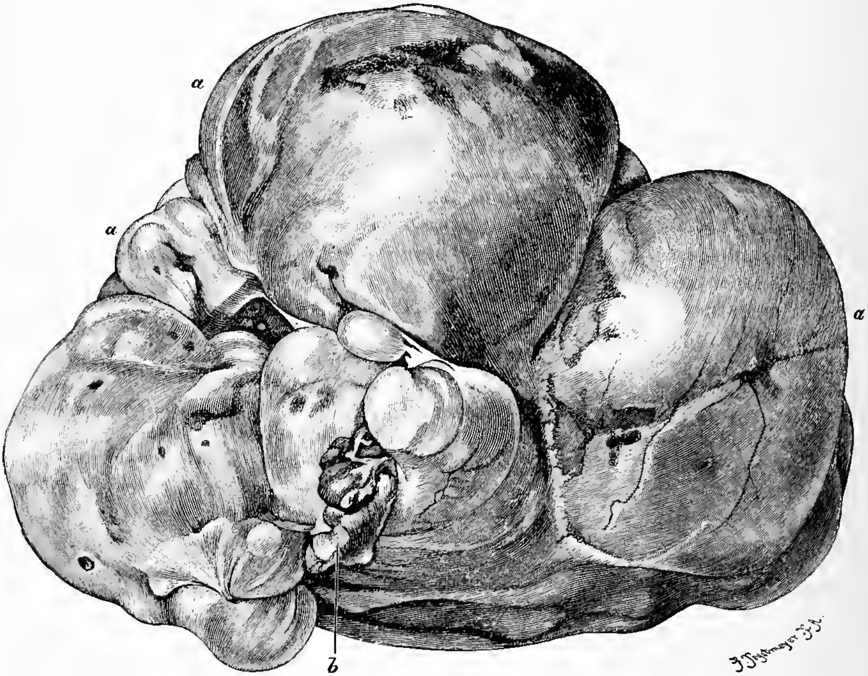

FIG. 507. OVARIAN CYST, PARTLY SIMPLE, PARTLY PAPILLIFEROUS.

(*Metastatic nodules in the peritoneum: reduced two-thirds*)

a cyst with smooth walls
b soft papillary outgrowth which has ruptured through the cyst-wall and is covered with simple columnar mucoid epithelium (metastatic nodules in the peritoneum)

317. By far the commonest of the **tumours** of the ovary is **cystoma** (ovarian colloid of VIRCHOW, myxoid cystoma of WALDEYER). Most cystomata are multilocular, a few are unilocular, and they differ from simple follicular cysts in that they are always associated with neoplastic proliferation, which may be either antecedent or subsequent to the appearance of cavities in the tumour. Their mode of origin shows that they belong to the group of epithelial new-growths, and one or both ovaries may be affected.

It is convenient to divide these growths into simple and papilliferous cystomata, the cyst-walls in the former being smooth, in the latter beset with papillary outgrowths.

Simple multilocular cystoma is a voluminous rounded tumour, usually lying free within the abdominal cavity but for its pediculate attachment, and weighing from 2 to 50 kilogrammes (Fig. 507). The tumour is lobulated, with a number of minor cysts (*a*) attached to it. In rare cases the mass is subdivided by

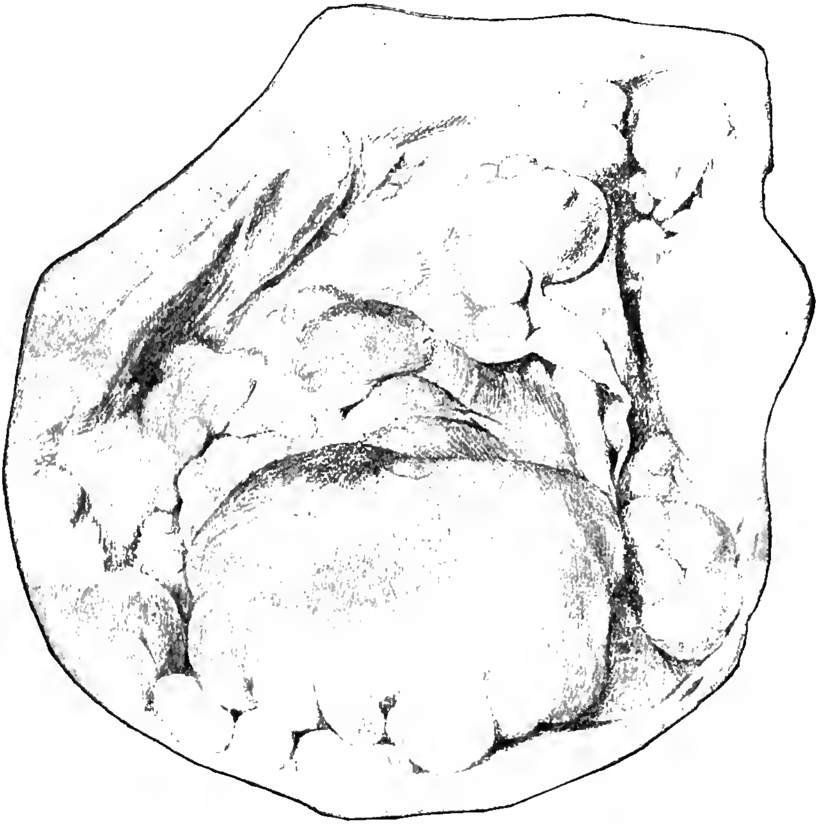

FIG. 508. SIMPLE MULTILOCULAR CYSTOMA.

(*Portion of the wall of the main cyst, showing numerous smaller cysts projecting into its cavity: five-sixths of the natural size*)

deep constrictions into several semi-detached portions. On section it is seen to consist of one or two main cysts about which are clustered a number of smaller ones of various sizes, with many lesser vesicles projecting inwards from their walls (Fig. 508). Now and then the mass of the tumour is made up of small aggregated cysts which give the section a honeycombed appearance (Fig. 509), and are interrupted here and there by cavities of larger size.

The smaller and middle-sized cysts usually contain a viscid mucus-like liquid, which is clear, or rendered somewhat turbid by the presence of white flakes, specks, and filaments, or occasionally whitish like catarrhal mucus, or stained reddish-brown or dirty-green by admixture with blood-pigment. The contents of larger cysts are in general more limpid, with little if any viscidity.

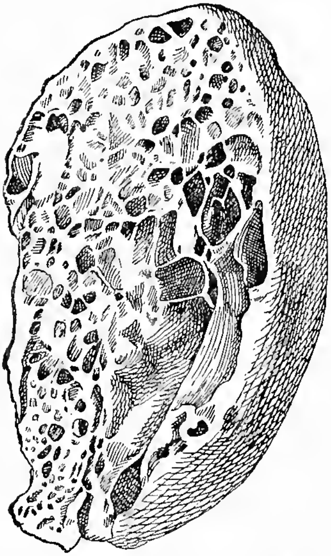

FIG. 509. PORTION OF A SIMPLE MULTILOCULAR CYSTOMA OF THE OVARY.

(*Five-sixths of the natural size*)

The walls of the cysts are usually composed of firm whitish fibrous tissue, and are often so thin that the contents shine through them. The thicker portions sometimes enclose small barely-visible cysts, or consist of spongy or adenoid tissue of a reddish or whitish colour, which yields mucus when it is cut into. The inner surface is smooth and glistening, and in a large cyst is often ridged with prominent spurs representing the remains of the septa formerly dividing the cavities of the constituent cysts that coalesced to form it.

The **papilliferous cystoma** may be either unilocular or multilocular (Fig. 510), and is characterised by the presence within its

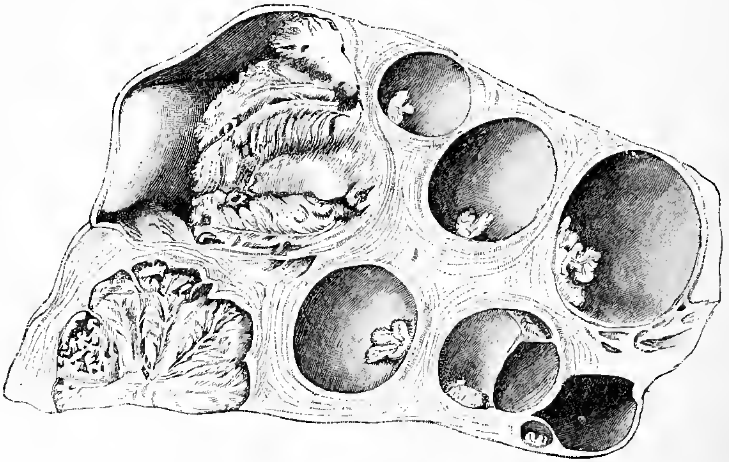

FIG. 510. SECTION THROUGH A PORTION OF A MULTILOCULAR PAPILLIFEROUS OVARIAN CYST.

(*Natural size*)

cavities of papillary excrescences of various sizes springing from the inner surface of their walls. When small they form merely roughnesses on the surface, or at most rise as little warty prominences; the larger ones are often so numerous and bulky that they fill up the cyst-cavity.

In other respects the tumour more or less resembles a simple multilocular cystoma, but the intercystic septa are usually thicker. When small it not infrequently consists of one or two cysts with a few papillary protuberances rising from the inner surface. Tumours in which the papillary outgrowths are exceptionally abundant have almost the appearance of a compact non-cystic neoplasm of somewhat soft texture.

Both the simple and the papilliferous varieties are in general lined with a single layer of flattened or tall columnar epithelium which is sometimes ciliated (Fig. 511 and Fig. 512 *c*), and shows signs of mucoid change such as typical goblet-cells. The contents of the cysts are evidently for the most part produced by this epithelium; and though no doubt local hyperaemic conditions may be followed by free transudation from the sub-epithelial blood-vessels, the epithelial mucous secretion forms an essential constituent, particularly in the case of the smaller cysts. Sometimes mucoid epithelial cells (Fig. 512 *d*) are discoverable in the cyst-contents, together with rounded cells of various sizes, free nuclei, oil-globules, fat-granule cells, red blood-corpuscles, and granular pigment.

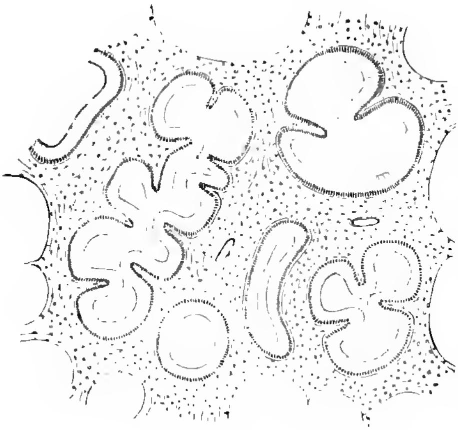

FIG. 511. PAPILLIFEROUS CYSTADENOMA OF THE OVARY.

(*Preparation hardened in Müller's fluid, stained with haematoxylin, and mounted in Canada balsam*: × 40)

The neoplastic structural elements from which the cyst developes are frequently demonstrable in the solid portions of the tumour as simple epithelial processes or gland-like tubules (Fig. 511). Both in simple and in papilliferous cysts, however, there is always a tendency to the elaboration of more complex structures, which may be regarded either as tubules whose epithelium is sacculated outwards, or as tubules indented by papillary ingrowths from the stroma (Fig. 511 and Fig. 512 *b*).

The first beginnings of the growth do not appear to be of the same nature in every case. Inasmuch as simple cysts containing papillary ingrowths are occasionally met with in the ovary, it would appear that the neoplastic proliferation may start in dilated follicles into which papillary processes intrude from the stroma. But as a number of gland-like tubules can generally be detected in the cyst-walls of multilocular tumours, and as these demonstrably furnish by their continued growth and multiplication the materials for new cysts, it is not improbable that the initial stage is the production of neoplastic epithelial tubules which have no prototype among the normal constituents of the ovary: on this view the growth would have to be regarded as a **cystadenoma**, the cystic excavation of its mass being a secondary feature. The tubules are derived from the epithelium of mature or residual embryonic follicles, or from the germinal epithelium of the ovary. It is probable that some developmental anomaly of the ovarian structure may furnish the starting-point of the tumour, inasmuch as in many cases its inception dates back to the age of childhood or even to the intra-uterine period (DORAN), while it is often bilateral, and its cysts are occasionally provided with ciliated epithelium.

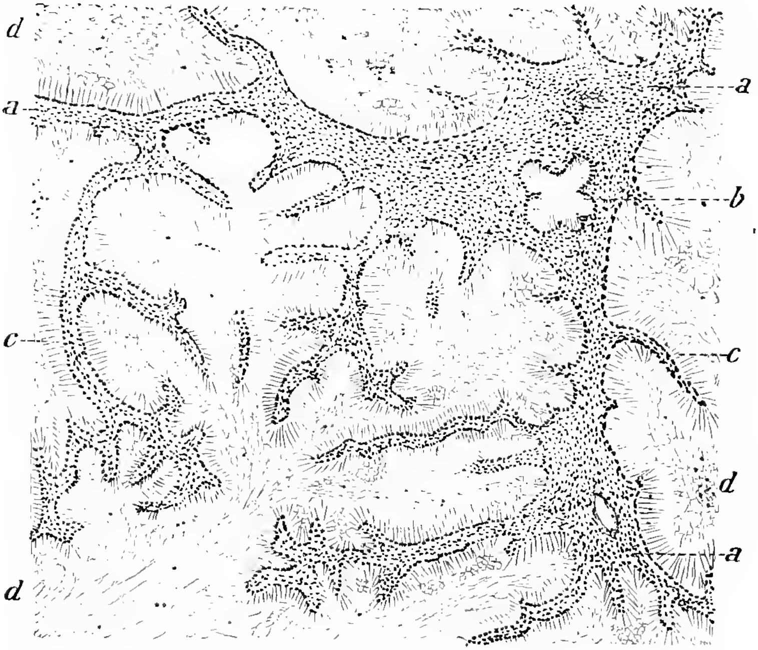

FIG. 512. PAPILLIFEROUS CYSTOMA OF THE OVARY.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin:* × 150)

a	stroma with papillary outgrowths	*c*	tall muciferous columnar epithelium
b	glandular tubule with small papillae	*d*	mucoid cells within the cysts

Cystoma of the ovary is generally a non-malignant growth, and though it sometimes reaches a very considerable size, and frequently becomes adherent to the parts around it, it does not produce metastases elsewhere. Forms are however met with that display a certain local malignancy (Fig. 507), inasmuch as they give rise to secondary growths, chiefly within the abdomen. Apparently such semi-malignant growths are always distinguished by the luxuriant development of the papillary ingrowths from the stroma (Fig. 513 *c d*) and of the epithelial structures (*b*) which they exhibit. It might almost indeed be laid down that the more abundant the papillary and epithelial overgrowth the more malignant is the tumour. The cysts accordingly tend to become entirely filled with ingrowing papillae, and on section the tissue looks almost continuous and acquires more and more the aspect of a marrowy cancerous growth. Not infrequently the papillae break through the cyst-wall and protrude from its surface as huge fungating or 'cauliflower' excrescences. Firm adhesions with contiguous organs are set up, and the growth spreads by degrees, mainly however in the broad ligament of the uterus. Now and again metastases are produced in the abdominal cavity, and

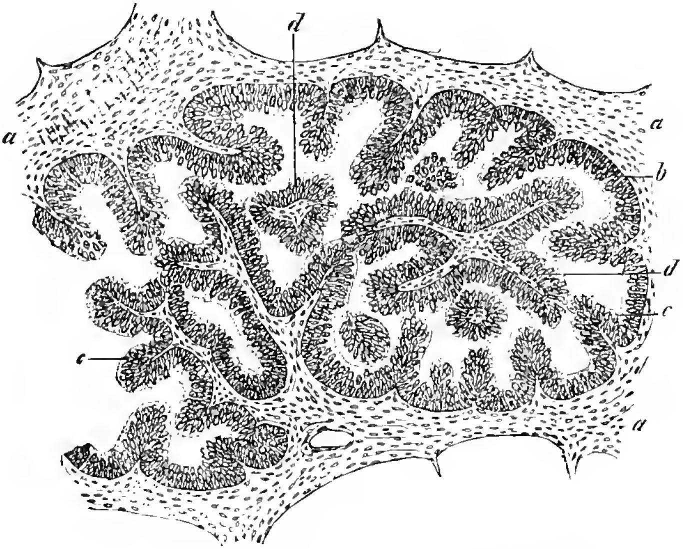

FIG. 513. PAPILLIFEROUS CYSTOMA OF THE OVARY.

(*Preparation hardened in Müller's fluid and alcohol, stained with alum-carmine, and mounted in Canada balsam:* × 120)

a stroma
b stratified epithelium
c longitudinal sections of papillary outgrowths
d transverse sections of papillary outgrowths

cases occur in which the peritoneum is studded at various places with papillomatous growths (Fig. 358). This feature of malignancy gives the tumour, from a clinical point of view, the characters of a carcinoma, and it is thus with some reason described as a **papilliferous cystocarcinoma** (*cystoma papilliferum carcinomatosum*). In some instances indeed the cancerous nature of the growth is more definitely suggested by the fact that the tissue from which the papillae spring is itself beset with nests of epithelial cells.

In rare cases **papillomata** make their appearance on the outer surface of the ovary, which have a general resemblance to the papillary ingrowths from the cyst-wall of a cystoma. When they are invested with proliferous many-layered epithelium, and give rise to peritoneal metastases, they may appropriately be described as examples of **papillomatous carcinoma**. If however their papillae possess only a single layer of epithelial cells they must be regarded as papillomatous **fibromata** or **adenomata**. Certain of the papillae are occasionally tipped with **cartilage**.

Calcareous concretions are constantly met with in the stroma of cystomata whose papilliferous character is strongly marked.

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318. Of the **solid tumours** of the ovary **carcinoma** is by far the commonest, though it is less common than cystoma. As a rule it takes the form of a rounded or slightly uneven tumour, which sometimes reaches the size of a man's head, but is usually smaller.

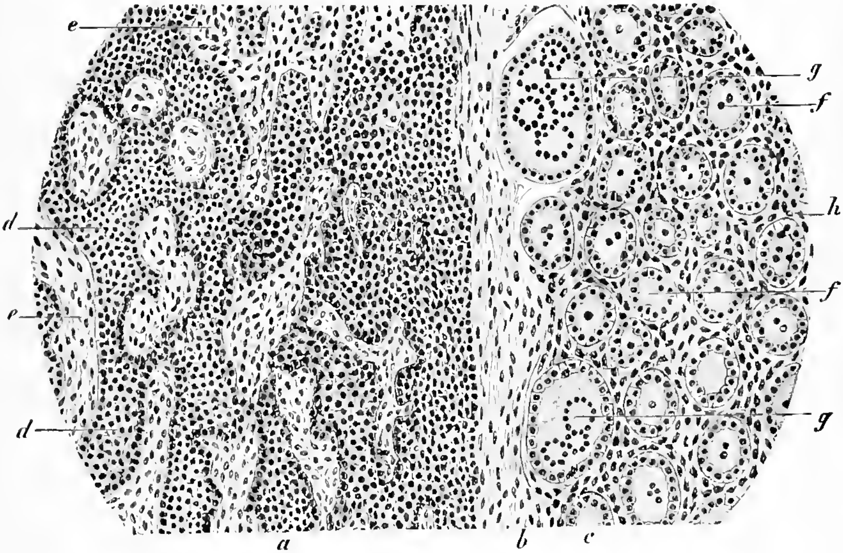

FIG. 514. CARCINOMA OF THE OVARY (VON KAHLDEN).

(Preparation hardened in Müller's fluid, and stained with haematoxylin and eosin: × 100)

a	tubular or gland-like structure	*d*	cellular trabeculae
e	alveolar structure	*f*	alveoli resembling primary follicles
b c h	fibrous stroma	*g*	alveoli with annular groups of nuclei

It occurs at all ages, even in youth, and on one or both sides. Some of these carcinomatous growths are firm, others are soft: some are glandular or adenomatous in structure (Fig. 514 *a*), others are alveolar (*f g*), and others again exhibit both types in combination. In very rare instances the growths contain nests of cells that very closely resemble ovarian follicles (*f g*), and they are then described as follicular adenomata. OLSHAUSEN, FLAISCHLEN, and others have met with gritty concretions like brain-sand in the cancerous cell-nests (psammocarcinoma). Some of the cancer-cells occasionally become mucoid; while in rare cases the stroma undergoes myxomatous metaplasia, and swelling up compresses the cellular masses into elongated trabeculae, which give a very peculiar texture to the substance of the growth. The ovarian follicles generally perish at an early stage, but isolated cysts are at times discoverable in the tumour; these probably existed before the new-growth began, and increased in size as it became larger.

All the forms of ovarian carcinoma give rise to metastases, the peritoneum being the usual seat of invasion.

Ovarian **fibroma, fibrosarcoma,** and **sarcoma** may be either unilateral or bilateral, and form rounded growths varying in size from that of a walnut to that of a man's head. They usually occupy the entire ovary, and as they grow retain the general shape of the organ. Their surface is more or less uneven, and they are generally firm in consistence, though soft and marrowy varieties are also met with.

In rare cases fibroma assumes the form of globular sharply-defined nodes, surrounded by normal ovarian tissue. According to ROKITANSKY, KLOB, KLEBS, and others, small fibromata may arise from corpora lutea, but this is doubtful.

When the tumour is small and the ovary accordingly not much enlarged, the substance of the growth sometimes encloses corpora fibrosa and follicles or their remains, in the form of small collections of epithelial cells. In rare instances patches of proliferous epithelium resembling adenoma occur in the tumour, which is then described as an **adenofibroma** or **adenosarcoma.** A fibromatous or sarcomatous tumour is moreover liable to be excavated into cysts, and thus the varieties known as **cystofibroma** and **cystosarcoma** are produced.

A few examples of haematangioma (MARKWALD), lymphangioma (LEOPOLD), and angiosarcoma (MARCHAND, ECKHARDT) have been recorded.

All the solid ovarian tumours are apt to undergo cystic excavation from fatty degeneration or internal softening. Fibromata may become partially calcified.

Dermoids of the ovary (Fig. 515), simple or compound, are not very rare. Simple dermoids are cysts that vary in size from that of a normal graafian follicle to that of a man's head, enclosing a characteristic greasy pulp and tufts of fair or reddish hair (*c*). Compound dermoid cysts contain, besides these, nodules of cartilage and plates of bone, teeth (*d*), and in exceptional cases structures belonging to the central nervous system or to the alimentary tract.

The corium or derma, which forms the fibrous portion of the wall of a dermoid cyst, may or may not be provided with papillae, and sometimes presents all the characters of the external skin; but when the wall is very thin the glandular structures and hair-follicles are usually absent. When teeth occur they usually protrude from the fibrous wall, their crowns being directed towards the interior of the cyst; sometimes they spring from bony plates.

The cyst is in general single, but two and even more have been observed in the same ovary. In a few cases both ovaries have contained dermoids.

Dermoids are not infrequently combined with adenocystomatous growths, forming what are called **mixed tumours**. The two kinds of cysts are either separate and distinct, or so combined that one and the same cavity is lined in different parts with stratified epidermal cells and with columnar epithelium, the latter being occasionally ciliated. The contents of the cyst are of a correspondingly heterogeneous kind. The wall sometimes encloses myxomatous and sarcomatous tissue.

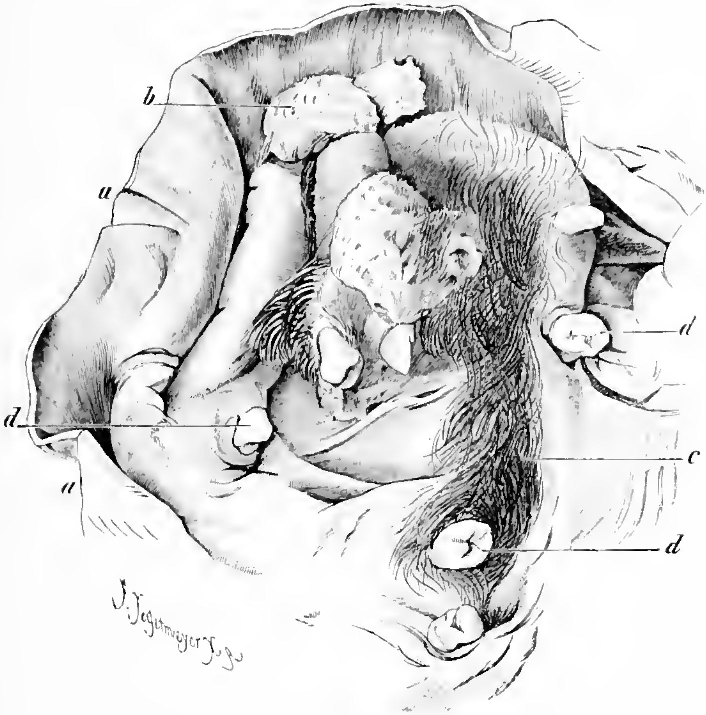

FIG. 515. DERMOID CYST OF THE OVARY.

a cyst-wall
b appendages composed of adipose and cutaneous tissues
c hair
d teeth

Dermoids grow very slowly, and do not usually give rise to any disturbance until adult life, that is to say until the ovaries are in a condition of functional activity. The cyst-wall often becomes inflamed, and adhesions to the surrounding structures are thus set up. Sometimes the cyst suppurates and becomes gangrenous, and rupture then takes place into some hollow viscus, such as the rectum, the vagina, or the bladder.

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CHAPTER CV

THE UTERUS AND FALLOPIAN TUBES

319. **Malformations.** The inferior segments of Müller's ducts become fused into a single canal between the eighth and the twelfth week of foetal life; in the fourth or fifth month the upper portion of this canal is transformed into the uterus and the lower into the vagina. The uterus then extends laterally towards the fallopian tube on either side in the form of two *cornua*, which are at a later stage merged again into the body of the organ.

When from any cause the fusion of the müllerian ducts is imperfect, the result is a series of anomalies of development whose common feature is the **duplication** of the genital canal in its uterine or its vaginal portion or both. Destruction or agenesis of the müllerian ducts on one or both sides gives rise to unilateral or bilateral aplastic **defects** of various degrees of gravity. Hypoplasia of the ducts or of portions of them leads to **rudimentary conditions** of the genital tract or of some of its parts.

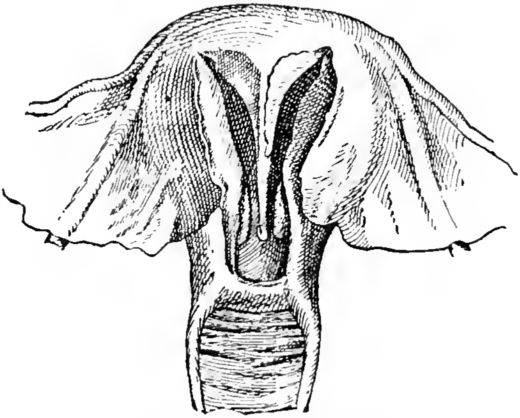

FIG. 516. UTERUS BILOCULARIS.
(*After* GRAVEL)

Two classes of malformations due to imperfect fusion of the müllerian ducts may be distinguished. In one of these the ducts unite into a canal that is externally single and normal in appearance, but is divided internally by a more or less complete longitudinal septum. When the cavity of the uterus is thus duplicated, the condition is termed *uterus bilocularis* or *uterus septus duplex* (Fig. 516), the analogous condition of the vagina being described as *vagina bilocularis* or *vagina septa.*

Incomplete subdivision, limited to the cervix and the external os or to the fundus, constitutes *uterus subseptus;* and the like partition of the vagina by a septum, which is sometimes perforated at a number of points, is known as *vagina subsepta.*

In the second class of cases the canals are not even externally united, but appear more or less widely divergent. The division is usually situated at the fundus of the uterus, which is thus either parted into two horns (*uterus bicornis*, Fig. 517) or assumes a form somewhat resembling that of an anvil (*uterus incudiformis*): cases however occur in which the division merely gives rise to a slight incurving of the middle of the fundus (*uterus arcuatus*).

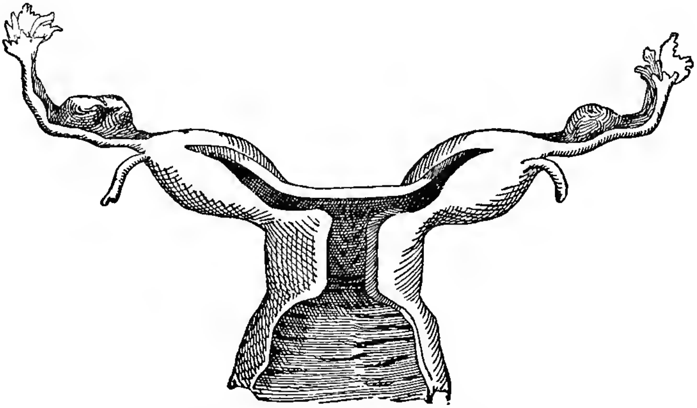

FIG. 517. UTERUS BICORNIS.
(*After* KUSSMAUL)

When a septum extends from the fundus of a bicornuate uterus to the external os, the condition is termed *uterus bicornis duplex* (Fig. 518), or if the septum is imperfect *uterus bicornis semiduplex.*

In rare instances the duplication is complete, extending throughout the uterus (*uterus bipartitus*), and sometimes throughout the entire genital canal (*uterus didelphys* and *uterus bicornis duplex separatus*): in this case the two uteri are generally stunted. Partial defects and general undergrowth are observed in cases where the uterus and vagina are single, as well as in cases of duplication.

In partial or total duplication, one of the canals may be occluded at some portion of its course (Fig. 518 *c*). Thus, in the case of a double vagina, one of the canals may be closed below, or end blindly in the middle or close to the uterus, owing to failure of development or to obliteration of the lower extremity of the müllerian duct on that side. One of the uteri or horns of a double organ is sometimes occluded: and in uterus bicornis one horn may be rudimentary and form a solid or a hollow process shut off from the uterine cavity, or may even be absent altogether (*uterus unicornis*). The tube on the undeveloped side is either normal, or rudimentary and shrunken to a mere string.

The slightest degree of unilateral arrest of development gives rise merely to oblique asymmetry of the uterus.

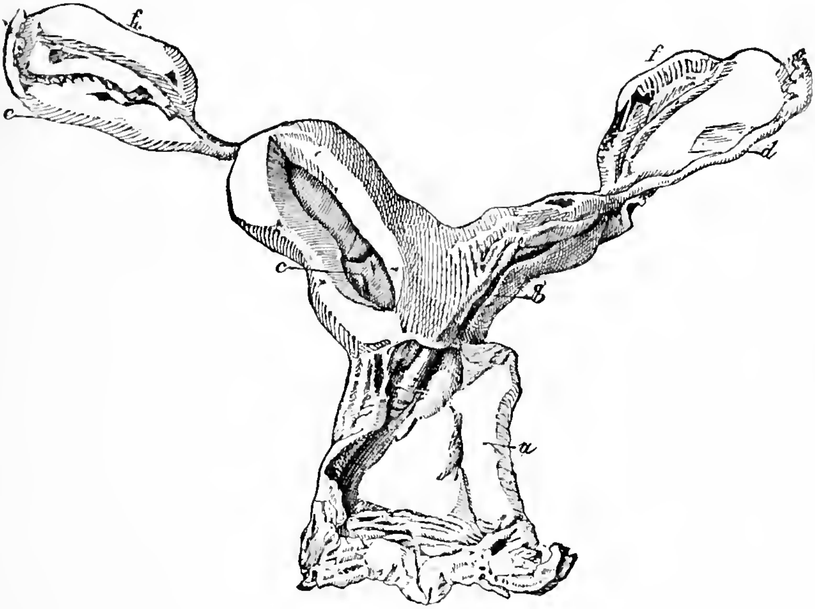

FIG. 518. UTERUS BICORNIS DUPLEX, WITH HAEMATOMETRA AND HAEMATOSALPINX.

(*From an unmarried woman aged 20: one-half the natural size*)

a	vagina	*d*	right tube
b	right uterus, with the os patent	*e*	left tube, distended with blood and ruptured
c	left uterus, with os occluded and cavity distended with blood	f f_1	ovaries

Even when the fusion of the müllerian ducts has taken place in normal fashion, the uterus may still suffer from local structural deficiency or general hypoplasia; in somewhat exceptional cases it is so stunted that it consists merely of a solid or a hollow muscular knob or cord.

The **rudimentary uterus** is generally bipartite or bicornuate, or appears as a mere cord connecting the two fallopian tubes. It is rare for the cornua to be absent while a middle portion corresponding to the body and the cervix persists: in such cases the tubes may be present or absent. Not infrequently the uterus begins by developing normally, but is arrested at a subsequent stage; and the body of the uterus, which after the sixth year should overtake and overpass the cervix in size, retains its foetal proportions or at least fails to attain its normal dimensions. This condition is variously described as *hypoplasia uteri*, *uterus foetalis*, or *uterus infantilis* (Fig. 519). The ovaries may notwithstanding be well developed.

Complete absence of the **fallopian tubes** is usually associated with the graver malformations of the uterus. Other anomalies are—separation of the tubes from the uterus, multiple abdominal ostia, conversion of the tubes into solid cords, atresia of the abdominal or of the uterine ostia, and stenosis or occlusion of the middle segment of one or both tubes.

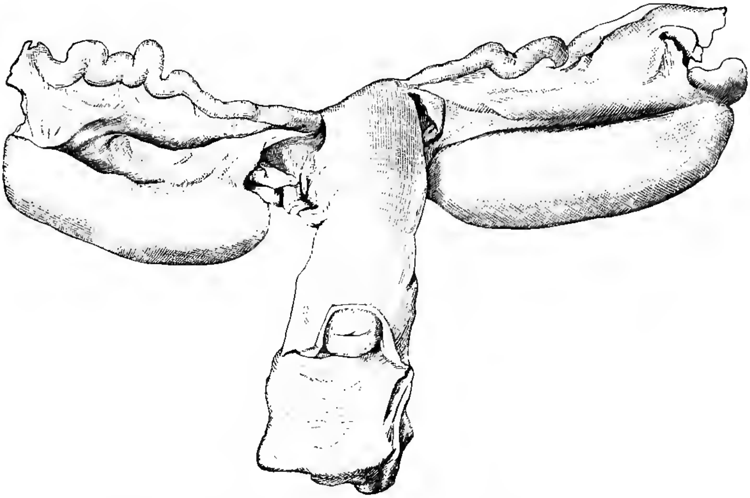

FIG. 519. INFANTILE UTERUS, WITH WELL-DEVELOPED OVARIES.
(*From a cretinoid dwarf aged* 18 : *five-sixths of the natural size*)

Partial or total absence of the **vagina**, with or without malformation of the uterus, is rare. More frequently some portion of the vagina is transformed into a solid cord.

Atresia and **stenosis** of the genital canal are most frequently met with along the course of the cervix down to its termination at the external os, and at the cervical and vulvar ends of the vagina. Cervical obstructions consist partly of mucous membrane and partly of muscular tissue. In the vagina the constriction may extend over the whole canal, or it may be only local and is then due to the presence of oblique or transverse folds or incomplete septa.

Stenoses of any considerable extent are usually associated with other abnormalities of the genital canal. When the uterus at puberty retains its infantile form, the vagina also remains short and narrow *(vagina infantilis)*.

Now and again a blind smooth-walled pouch is met with in the posterior wall of the vagina, immediately behind the vulvar entrance: it runs parallel to the vagina, or diverges from it laterally, and is sometimes large enough to admit the finger. According to Breisky, this pouch is produced by the dilatation and elongation of a hiatus in the mucous membrane that is normally present in that situation.

The normal **hymen** is a fold of mucous membrane springing from the posterior vaginal wall, which originally separates the vagina from the urogenital sinus, and after the sinus closes forms a border round the vertical cleft at the vaginal orifice.

In new-born infants the hymen is tubular, and it sometimes retains this form in later life. Normally however it is of a semilunar shape, though it is occasionally annular, and by no means rarely imperforate (*atresia vaginae hymenalis*). It may be double (*hymen septus*), or perforated by several small openings (*hymen cribriformis*); its free margin may be dentate or furnished with a papillary fringe (*hymen denticulatus vel fimbriatus*); or there may be a second hymen above the first one. Congenital absence of the hymen is very rare. Cysts of the hymen have occasionally been observed.

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320. The **fallopian tubes** or **oviducts** are muscular tubes lined with a mucous membrane whose epithelium consists of a single layer of ciliated columnar cells, and which is arranged in numerous longitudinal folds and arborescent processes.

Congestion of the mucous membrane is liable to occur in connexion with the general hyperaemia of the genital system accompanying menstruation, coition, and many of the acute infective diseases: it is sometimes so extreme as to give rise to haemorrhage, and in exceptional instances to the effusion of blood from the tubes into the abdominal cavity.

Inflammation of the tubes, or **salpingitis**, is most commonly an after-effect of inflammation of the uterus or pelvic peritoneum; when it is of uterine origin it is nearly always followed by pelvic peritonitis and superficial inflammation of the ovary. The resulting exudation causes the tubes to become adherent to the adjacent structures, such as the ovary, broad ligament, or posterior wall of the uterus; and in the course of time the adhesions are converted into more or less firm membranes and bands of fibrous tissue (Fig. 504 c c_1 and Fig. 520 e), which as they contract drag the tubes from their normal position, while the abdominal ostia often become impervious.

Salpingitis is distinguished as mucous, purulent, muco-purulent, serous, or haemorrhagic according to the nature of the exudation contained in the tubes. When the abdominal ostium is closed by adhesions and the uterine extremity of the tube is at the same time obstructed by swelling of the mucous membrane, by inspissated secretion, or by abrupt flexure or constriction of the channel, the liquid exudation whatever be its nature accumulates in considerable quantity within the abstricted portion, and gives rise to the conditions known as **hydrosalpinx**, **pyosalpinx**, and **haematosalpinx** (Fig. 520 c).

When the collection of liquid is moderate in amount, the tube appears dilated and tortuous (Fig. 504 c c_1); when it is large, the tube is distended into a thin-walled spheroidal sac (Fig. 520 c). Its wall is apt to suppurate in places if the inflammation is purulent, and rupturing allows pus to escape into the abdominal cavity. In other cases the pus becomes inspissated and calcareous. Rupture occasionally takes place also when the tubal contents are mucous or bloody. If the uterine ostium is of normal width or merely contracted, the accumulated liquid may escape from time to time into the uterus (*hydrops tubae profluens*). Under certain special conditions, haemorrhagic, mucous, or purulent liquid retained in the uterine cavity regurgitates through the uterine ostium, and so gains access to the peritoneal cavity through the abdominal ostium, or if this is occluded accumulates in the tube itself.

Salpingitis is induced by various kinds of infection, the purulent forms being due to the ordinary pyogenic micrococci, to *Gonococcus*, or more rarely to *Pneumococcus*, though when the tubes come under examination the pathogenic micro-organisms are often no longer to be discovered. The mucosa is more or less densely infiltrated, and the infiltration sometimes extends to the muscular and serous coats. In chronic cases the folds and processes of the mucous membrane often become hyperplastic, and the other constituents of the tube-wall are also apt to be thickened; the affection may then be described as **proliferous salpingitis.** The mutual cohesion of internal folds and processes that have been partially denuded of epithelium produces small isolated cysts with an epithelial lining. In rare instances the wall of the uterine end of the tube is beset with tumour-like thickenings that may be as large as a bean; they are due chiefly (CHIARI) to hypertrophy of the muscular coat, and enclose little epithelial cysts. CHIARI attributes their formation to invaginations of the mucous membrane, followed by hypertrophy of the muscular layer, and regards them as evidence of antecedent inflammation.

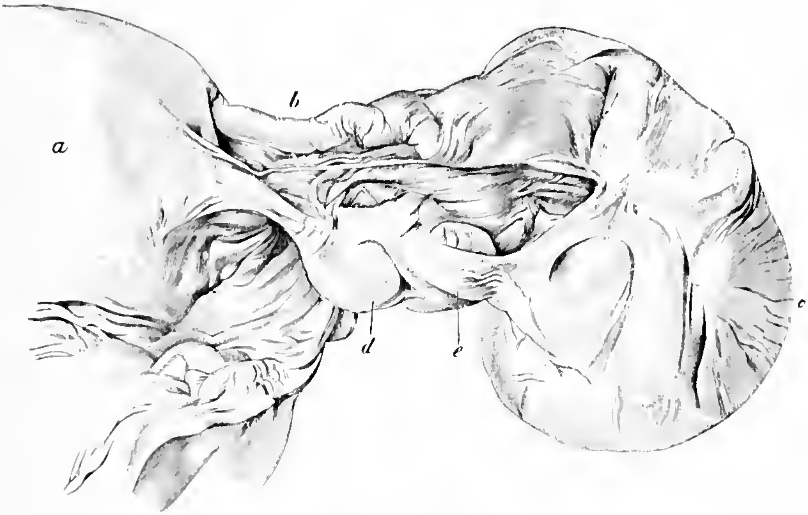

FIG. 520. HYDROSALPINX, WITH ADHESIONS OF THE TUBE AND OVARY.
(*Two-thirds of the natural size*)

a uterus
b uterine extremity of the tube
c abdominal extremity of the tube, dilated and adherent to the surrounding structures
d ovary
e membranous adhesion

Tuberculosis of the tubes makes its appearance in children as well as in adults, and is usually bilateral. As a rule, the uterus is subsequently infected, though it is sometimes the primary seat of the disease. The initiation and extension of the tuberculous process take place in the same way as in other mucous membranes, the course of the affection closely resembling that of tuberculous ureteritis. In chronic cases that are well advanced the tubes are dilated and filled with caseous matter, the mucous membrane, and occasionally the muscular coat also, being transformed into grey gelatinous granulation-tissue whose surface is in process of caseous disintegration. In some cases the tubal affection is the only tuberculous lesion in the body. As the disease progresses it is apt to induce peritonitis, which is sometimes purulent or even gangrenous in character, owing to multiple or mixed infection.

Among the **tumours** met with in the tubes fibroma and fibromyoma are the chief. They appear as small nodules arising from the muscular coat, but are much rarer in the tubes than in the uterus. Local overgrowth of the mucous processes and folds gives rise to papillomatous fibroma. Carcinoma occasionally extends to the tubes from the uterus or ovaries: in very rare cases it appears in the tubes as a primary growth.

Cysts are at times developed on the external surface of the tubes, the commonest kind being derived from the hydatid of Morgagni, a pedunculated vesicle situated at the abdominal end, and representing the uppermost portion of the müllerian duct. Small cysts with colloid contents are often observed on the tubes and broad ligaments; they vary from the size of a poppy-seed to that of a pea, and probably originate in portions of the paroöphoron or epoöphoron.

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321. The **uterus** is an elongated pyriform muscular organ, flattened from before backwards; in the nullipara it measures from 5·5 to 8 centimetres in length, in the primipara and multipara it is from 9 to 9·5 centimetres long and from 3·5 to 5·6 centimetres broad. It is largely made up of unstriped muscular tissue, and encloses a cavity lined with a mucous membrane (endometrium) that is abundantly provided with glands. With the tubes, which extend laterally from its fundus, it is interposed between bladder and rectum, and raises up the peritoneum, reflected over it from the bladder in front and the rectum behind, into a transverse fold which divides the vesico-uterine from the recto-uterine pouch.

In children and virgins the uterus is applied to the posterior wall of the bladder, and retains this relation even when the bladder is empty, the recto-uterine pouch being thereby opened out. In women who have borne children the uterus is sometimes in contact with the posterior wall of the bladder, and sometimes turns away from it at a right angle towards the recto-uterine pouch. Gynaecologists regard the former as the normal position.

The axis of the body of the uterus usually forms with the cervix an obtuse angle re-entrant anteriorly, a position described as that of **anteflexion**. A slight degree of anteflexion is physiological; when the flexion is abrupt and considerable the condition must be regarded as morbid (Fig. 521).

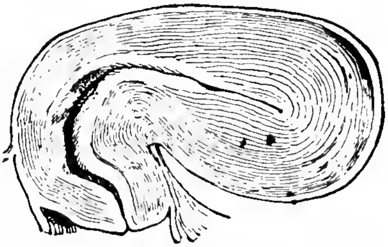

FIG. 521. ANTEFLEXION OF THE VIRGIN UTERUS.
(*Sagittal section: five-sixths of the natural size*)

When the angle formed by the uterine with the cervical axis is re-entrant posteriorly the uterus is said to be in a position of **retroflexion** (Fig. 522). In some case the uterus, while retaining its normal angular relation to the cervix, is rotated as a whole forwards or backwards to an abnormal extent, the result being **anteversion** or **retroversion** as the case may be.

Versions and flexions of the uterus are very frequently intercombined, and are occasionally complicated by lateral deviation or general displacement. Extreme flexion may make the utero-cervical angle acute.

Versions are due chiefly to enlargement of the uterus, and also to repeated abdominal straining, when the substance of the organ is firm and unyielding: should the uterus be soft and flabby, the same forces will give rise to flexion. In rare instances morbid anteflexion or retroflexion is dependent on some developmental anomaly. Adhesions of the uterus to the surrounding structures are also capable of inducing version or flexion.

The principal effects of flexion are retention of the menses, venous engorgement, and haemorrhage into the mucous membrane.

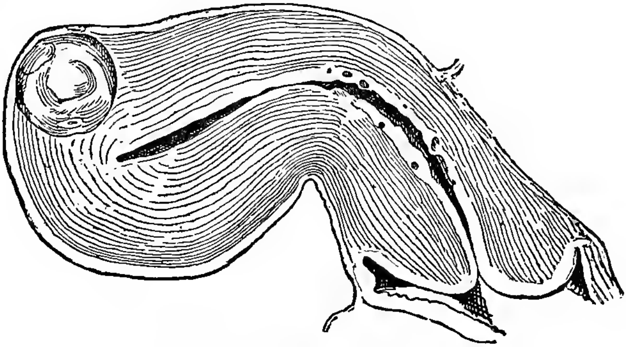

FIG. 522. RETROFLEXED UTERUS.

(*From a married woman with a small interstitial fibroma: five-sixths of the natural size*)

Lateral deviation (version or flexion) is brought about by tumours, parametritic exudations, adhesions to the surrounding parts, and the like.

Prolapse and *procidentia uteri* are terms used to designate the downward displacement of the uterus as a whole towards the vagina, whereby the os approaches the vulva and at length protrudes from it (Fig. 523 *c*), the descent being rendered possible by the relaxation of the structures that normally retain the uterus in its proper position.

In the simplest cases of this displacement the uterus lies lower in the pelvis than normal, but the os is not visible at the vulva. In what is called incomplete prolapse, part of the uterus protrudes through the vulvar orifice; and in complete prolapse the entire uterus (*c b*) lies in front of and outside the pudenda and is covered by the inverted vagina. The utero-cervical axis of the prolapsed organ is in some cases straight, while in others it is flexed (*c b*).

The tissue of the inverted vagina ultimately becomes more or less hypertrophied, its rugae are effaced, and its epithelial lining dries and assumes the appearance of horny epidermis. Inflammation and ulceration take place in the exposed parts as a result of abrasions and other injuries, and the uterus and cervix become engorged and swollen, and in the end hypertrophic. The traction exerted on the protruding cervix by the vagina drags open the os uteri, and the lower portion of the cervical canal is widely everted (*ectropion*): in this way the internal os occasionally comes to be visible at the vulva. In other cases again the external os is completely occluded.

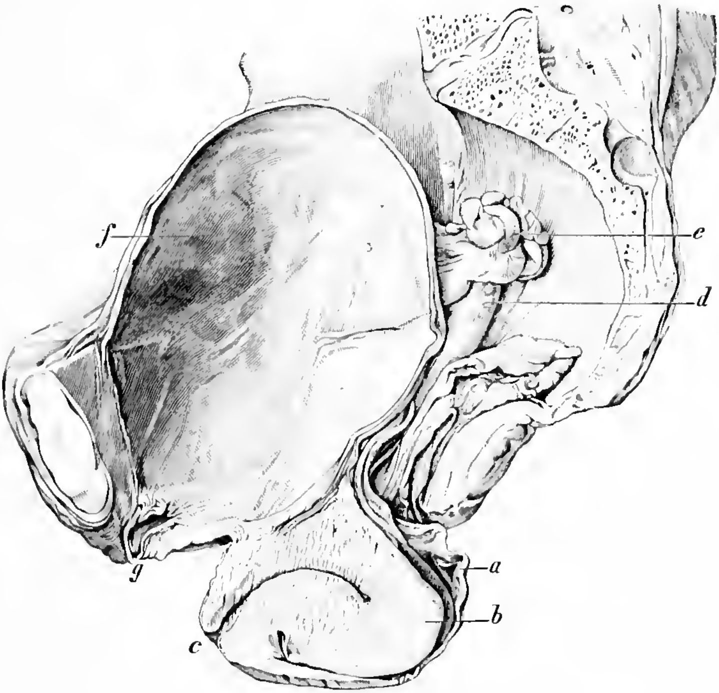

FIG. 523. PROLAPSE OF THE UTERUS.

(*Sagittal section through the pelvis: one-third of the natural size*)

a inverted vagina
b prolapsed and retroflexed uterus
c external os
d ovary
e abdominal end of the tube
f bladder
g urethra

The prolapsed uterus drags down the anterior wall of the rectum and the posterior wall of the bladder, and so gives rise to sacculation (rectocele and cystocele) of these viscera (Fig. 523).

By adhesions with the pelvis or in the parts about the uterus the displaced organ may become so fixed in its false position that it can no longer be replaced.

Elevation or upward displacement of the uterus results from the growth and upward pressure of tumours beneath it, or from the traction of supra-uterine tumours connected with the uterine wall as they rise out of the pelvis. In certain cases the contraction of peritonitic (perimetritic) adhesions has a like effect in raising the uterus. The effect is to elongate the uterus and vagina very considerably, until in some instances the vaginal fornices are effaced and the funnel-shaped vagina leads straight into the external os.

Inversion is the invagination of the fundus of the uterus into its cavity, by which the organ tends to be turned inside out. In slight degrees of this affection the fundus is still above the external os; in more extreme cases the fundus enters the vagina, and finally protrudes through the vulva. The three degrees are distinguished as invagination of the fundus or partial inversion, complete inversion, and prolapse of the inverted uterus.

Apart from cases arising in connexion with parturition, inversion is brought about by the growth of intra-uterine tumours, which dilate the uterus, weaken its wall, and at the same time exert traction upon the fundus. As a rule the inverted fundus proceeds no farther than the cervix, where it is grasped and retained. The condition is far from common.

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322. The **mucous membrane** (endometrium) of the uterus is a structure abounding in glands, which owing to the absence of a distinct submucosa is directly continuous with the connective tissue of the muscular wall. Its surface is covered with tall columnar and ciliated epithelium, which passes down into the deeper glands. Its stroma is composed of slender fibres enclosing a multitude of cells, the deepest layers being pervaded by muscular fasciculi. The glands consist of simple and branching tubules lined with columnar epithelium, some of them being convoluted at their deeper extremity. In the cervix the mucous membrane is disposed in ridge-like folds and elevations described as the *arbor vitae* or *plicae palmatae*. At the lower part of the cervix the mucosa is firmer and more fibrous, while the glands become less numerous and some of them are shorter than those in the body of the uterus: they are often sacculated and racemose. About the lower third of the cervical canal the columnar epithelium passes into the stratified squamous epithelium which covers the lips of the external os, and is continuous with the squamous epithelium of the vagina. At the part where the plicae palmatae come to an end, small conical papillae make their appearance, and become particularly abundant as the external os is approached.

The number, size, and conformation of the glands, and the extension downwards of the columnar epithelium, vary somewhat in different women, and in the same woman at different times. In many the vaginal portion is highly glandular, while in others it is almost or altogether devoid of glands, consisting essentially of vascular connective tissue and muscular fasciculi.

In old age the endometrium generally diminishes in thickness: it becomes less cellular and more distinctly fibrous, the epithelial cells become shorter and lose their cilia, and some of the glands, particularly those of the cervix, are obliterated, while those that remain are dilated into little cysts (*ovula Nabothi*).

During **menstruation** the mucous membrane is the seat of intense congestive hyperaemia, and a certain quantity of blood is extravasated from the superficial vessels of the mucous membrane. Some of the extravasated blood escapes between the epithelial cells, but as a rule portions of the surface epithelium are separated and disintegrate. In places where the extravasation is considerable, the superficial connective layers are also exfoliated, and continuous tracts of mucous membrane may thus be removed and lost. In the intermenstrual period the loss is made good by epithelial proliferation.

The quantity of blood lost at the catamenia varies greatly in different women, and is sometimes enormous, the latter condition being described as **menorrhagia**. In certain cases from the second to the fourth day of menstruation not only blood but shreds and continuous portions of the mucous membrane are extruded, with or without pain, the exfoliated membranes being occasionally tube-like or even presenting the appearance of complete casts of the uterine cavity. This condition is usually described as **membranous dysmenorrhoea.** The surface of the exfoliated membrane may be smooth or ragged; sometimes it is smooth on one side and rough on the other.

Its composition differs in different cases. At times the cast is made up entirely of fibrin, round lymphoid cells, and red blood-corpuscles, and is then simply a membranous coagulum which has been formed on the surface of the uterine mucous membrane. In some cases the coagulum is so firm that it must have been formed during the preceding menstrual period, being exfoliated only when fresh haemorrhage took place beneath it.

In other cases the extruded membrane is unmistakably a portion of the uterine mucosa, as it consists of soft fibro-cellular tissue, with blood-vessels and glandular tubules, whose epithelium is in part normal and in part swollen and desquamating. Portions of the tissue are moreover densely infiltrated with red blood-corpuscles. In such instances it is plain that a portion of the endometrium, infiltrated with menstrual blood, has been exfoliated *en masse*, and even the deeper layers of the membrane may be thus detached and extruded.

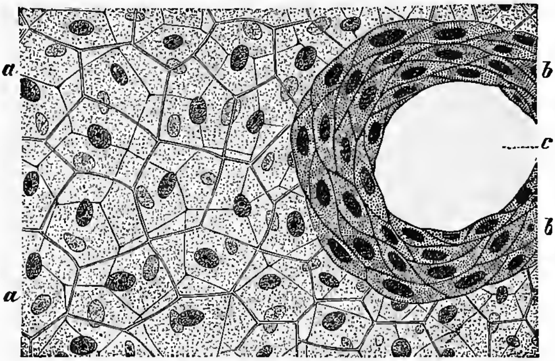

FIG. 524. MEMBRANOUS DYSMENORRHOEA.

(*Membrane extruded through the vagina: preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 300)

a twofold layer of polygonal squamous epithelial cells

b concentric arrangement of the cells round the mouth of a gland

c lumen of the gland-tube

In a third group of cases the membrane is composed of one or more layers (Fig. 524 *a* and Fig. 525 *c*) of polygonal squamous epithelial cells, with lacunae at regular intervals corresponding to the mouths of the uterine glands (Fig. 524 *c* and Fig. 525 *b*), about which the epithelium is concentrated into a broad ring (Fig. 524 *b*).

Inasmuch as the normal uterine mucous membrane contains no squamous epithelium, while the vaginal epithelium though squamous contains but few glands, these membranes must in general be derived from the cervical canal. But as some of them measure from 3 to 4 centimetres in length, it must be assumed that in these instances the squamous epithelium extends unusually far into the cervix, or is even continued into the body of the uterus. According to ZELLER, stratified squamous epithelium is often produced in the cervix and in the body of the uterus in cases of chronic endometritis.

When the membranes consist solely of squamous epithelium uninterrupted by glandular lacunae, they may be derived from the vaginal portion of the uterus, or from the vagina itself.

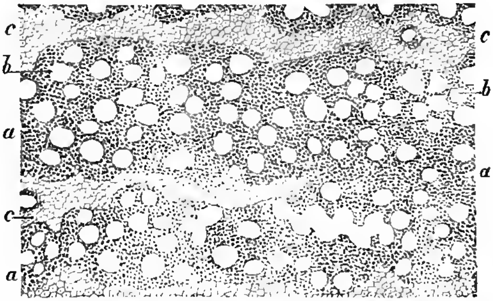

FIG. 525. MEMBRANOUS DYSMENORRHOEA.

(*Membrane extruded through the vagina: preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam:* × 30)

a layer of stratified epithelium
b mouths of the glands
c layer of polygonal cells

Metrorrhagia is the term applied to haemorrhage from the uterine mucous membrane at some time other than during the menses, gestation, or the puerperal period; it is associated with haemophilia, scurvy, acute inflammations, various infective diseases such as typhoid fever, small-pox, scarlet fever, measles, cholera, etc., and with toxic conditions, such as phosphorus-poisoning. Metrorrhagia is moreover often due to hyperplastic overgrowth of the uterine mucous membrane, to ulcers, and to tumours of the endometrium or underlying uterine parenchyma. When the haemorrhage is copious, the blood sometimes coagulates within the uterine cavity.

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323. **Endometritis**, or inflammation of the mucous membrane of the uterus, may be limited to the cervix or to the body of the uterus, or it may involve the entire internal surface of the organ. As a rule it is consecutive to inflammation of the vagina, but in exceptional cases it is of haematogenous origin and associated with menstruation or the puerperal state. The infective varieties are in most cases due to the action of the *Gonococcus* of Neisser.

Acute endometritis, while it corresponds in regard to its course and general characters with the inflammations of other mucous membranes, exhibits certain peculiar features. The inflammation is usually of a catarrhal nature.

The normal secretion of the cervical glands is viscid and slimy; that of the uterine glands is more limpid inasmuch as it contains less mucin, and it is scanty in amount. In catarrhal endometritis the uterine secretion is profuse, and becomes slimy or even purulent (leucorrhoea or *fluor albus*), in which case it frequently contains micro-organisms (Küstner). In severe purulent inflammation blood is sometimes mingled with the pus.

In recent catarrh the mucous membrane is red, swollen, sodden, and infiltrated with cells or with extravasated blood. Portions of the mucous membrane occasionally undergo suppuration or become exfoliated, if the inflammation is sufficiently intense (exfoliative endometritis).

Chronic endometritis leads to grey pigmentation and atrophy of the mucous membrane, affecting in particular the glands, some of which are obliterated. The fibrous tissue also is liable to become less cellular and firmer, but not by any means in all cases, the endometrium having great regenerative powers.

The atrophic mucous membrane is sometimes smooth and covered with low epithelial cells destitute of cilia. It not infrequently encloses small cysts, or is somewhat uneven and corrugated, or beset here and there with small polypous or papillary excrescences. These changes have led to the affection being described as **hyperplastic endometritis** (fungous, villous, or polypous endometritis). The corrugations are however in some cases due merely to the unequal distribution of the atrophy; in other cases within the prominent portions the fibrous tissue or the glands are proliferous.

While the inflammation continues the hyperplastic fibrous tissue is infiltrated with leucocytes. After it has subsided the infiltration disappears, and the tissue looks normal or is at most a little firmer; it often continues to be highly vascular.

Small **cysts of retention** (*ovula Nabothi*) are frequently formed by the obstruction of the openings of the glands in the cervical mucous membrane. The secretion which accumulates in them consists of colourless or white and turbid mucus, or of pus, according to the nature of the initial inflammation. Where the cervical mucous membrane is soft and cellular, and covered with columnar epithelium, the little cysts project above the surface; where the tissue is firmer and covered with stratified squamous epithelium, as for example about the lips of the os uteri, the cysts lie deeper.

As the catarrhal inflammation subsides the contents of the cysts usually become inspissated into a whitish pulp; but many of them persist for an indefinite time, so that they are very often met with even long after the inflammation has ceased. Glandular cysts occasionally make their appearance independently of any catarrh of the mucous membrane.

Not infrequently the cystic dilatation of the glands is accompanied by hyperplasia of the mucosa, which leads to permanent corrugation or papillary overgrowth of the folds that contain the cysts. According to ZELLER, chronic endometritis in the cervix or the body of the uterus sometimes results in the production of new stratified squamous epithelium, and the polypous excrescences arising from these parts are not infrequently invested with squamous cells.

When the columnar epithelium extends downwards as far as the vaginal portion, a condition sometimes observed in women who have never suffered from endometritis (FISCHEL, KÜSTNER), the external os appears to be surrounded by a bright-red border of varying width, which is in sharp contrast with the somewhat livider tint of the rest of the neck. When the lips of the external os are patulous, owing to laceration produced during a previous labour, part of the cervical mucous membrane is everted and becomes visible from the vagina. Cervical inflammation also, by inducing a swollen condition of the vaginal portion, is apt to give rise to more or less extensive eversion of the reddened mucous membrane of the cervix near the os. Such eversions are sometimes described by the term *ectropion*.

Whether endometritis is associated with ectropion or not, the stratified epithelium about the os is liable to become macerated and desquamated (Fig. 526 *d*) under the action of the morbid secretions escaping from the cervix. It is probable also that small epithelial vesicles are produced in the course of inflammations of the vaginal portion that are accompanied by profuse exudation, their genesis and structure being similar to those of the epidermal vesicles formed in certain inflammations of the skin. Like these they discharge their contents when the film that covers them gives way. By one or other method **erosions** may be produced in the epithelium, which are either made good at once by regenerative proliferation, or persist for a time as more or less denuded patches.

The exposed fibrous tissue, whether actually denuded or covered only by fragile epithelial cells, is in general intensely red, bleeds easily, and is more or less densely infiltrated with leucocytes (*d*). When owing to the loss of surface epithelium the mouths of the glands are wide and gaping (*c*) the os assumes a very uneven appearance. In exceptional cases the exposed fibrous tissue disintegrates at certain points and minute **ulcers** are thus produced.

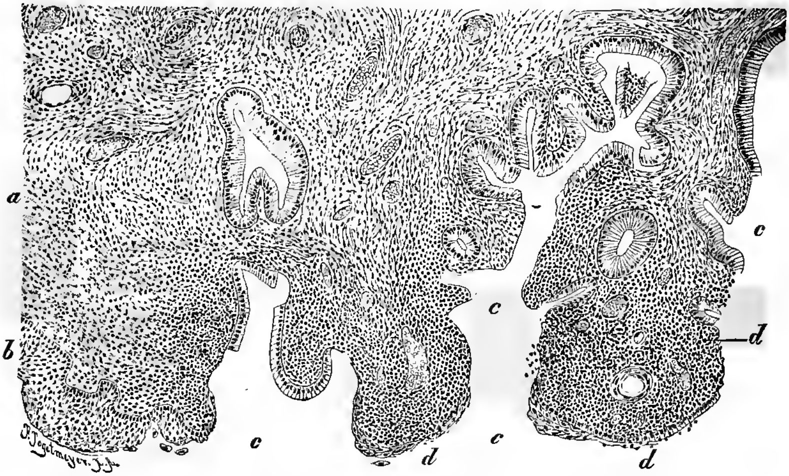

FIG. 526. PAPILLARY EROSION OF THE VAGINAL PORTION OF THE UTERUS.
(*Preparation hardened in Müller's fluid, and stained with haematoxylin* : × 45)

a connective tissue of the vaginal portion
b stratified squamous epithelium
c open mouths of dilated glands
d proliferous tissue infiltrated with cells

When an erosion does not heal up by the re-covering of its surface with squamous epithelium, the denuded tissue beneath is usually transformed into highly-cellular germinal tissue (*d*). Even when the epithelial covering is renewed, it consists in most instances of but a single layer of cells.

In chronic cases the interglandular tissue often becomes hyperplastic, and rises in papillary or villous protuberances, the resulting condition being termed papillary erosion. Not infrequently also small cysts are formed in the eroded parts, owing to the accumulation of retained secretion within the glands; this is sometimes described as follicular erosion. When the cysts break through to the surface, they remain as pits or excavations which are described as follicular ulcers.

If the inflamed condition of the endometrium is removed by appropriate treatment, and the irritation of the cervix maintained by the uterine discharge comes thus to an end, the bright-red mucous membrane gradually pales, and is recovered with new stratified squamous epithelium (KÜSTNER) growing inwards from the periphery, so that at length the parts resume their normal appearance.

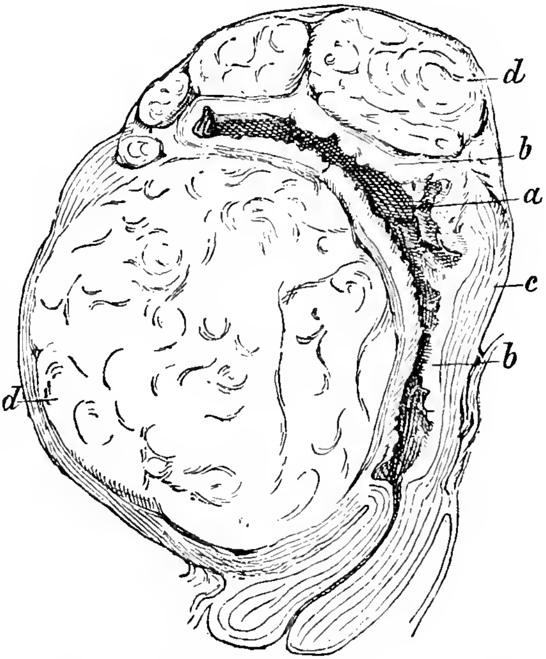

FIG. 527. TUBERCULOSIS, PYOMETRA, AND MULTIPLE FIBROIDS OF THE UTERUS.

(*Sagittal section: five-sixths of the natural size*)

a elongated uterine cavity
b caseous tissue
c uterine wall
d fibroids

Though minor erosions and ulcers are occasionally produced at the external os in some of the ways above referred to, it must be kept in mind that the great majority of so-called 'ulcerations' are nothing more than everted patches of cervical endometrium beset with papillary excrescences. Each of these is completely covered with epithelium of some sort, usually cubical, and there is no real breach of continuity in the mucous surface; while what appear to be granulations are simply villous projections due to local hyperplasia. These projections are separated by glands or gland-like pits, and when they become coherent they enclose cyst-like cavities.

Croupous and **diphtheritic inflammations** of the uterine mucous membrane are very rarely met with, except in the puerperal state. When they occur it is usually in connexion with typhoid fever, cholera, scarlet fever, or small-pox, or in the parts adjacent to foul carcinomatous ulcers. Gangrenous endometritis is a somewhat rare concomitant of puerperal septic pyaemia.

Tuberculosis of the endometrium may either be a primary lesion (Fig. 527), or be due to secondary extension from a tuberculous tube. It begins with the formation of small granulomatous nodules that rapidly break down by ulceration. In advanced tuberculosis the whole inner surface of the uterus is transformed into a single large ulcer, which is overlaid with caseous granulations (*b*), and smeared with caseous pus and pulpy detritus. Small grey or yellow tubercles are sometimes visible in the reddened vaginal portion of the uterus or in the vagina.

Syphilitic infiltrations and **chancres** are at times observed on the vaginal portion, but they are not common. When present they have much the same appearance and characters as syphilitic ulcers of the skin or the mouth.

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324. **Hyperplastic growths** of the uterine mucous membrane appear as an effect of chronic inflammatory processes, and in connexion with pregnancy and with menstrual disorders.

Such overgrowths in the vaginal portion usually consist of firm fibrous tissue, infiltrated in places with leucocytes. The cervical papillae sometimes grow out into warty structures analogous to the inflammatory papillomata (*condylomata acuminata*) of the skin. Like these they are generally associated with gonorrhoeal infection, although they are now and then produced by other kinds of irritation. When large they assume the form of 'cauliflower-excrescences,' their arborescent papillae being covered with thick layers of stratified squamous epithelium.

The hyperplastic growths of the endometrium invariably consist of soft fibro-cellular tissue like that from which they spring, and are characterised by the presence in them of typical uterine glands (Fig. 528 *a*) in varying numbers. When these glands are normally numerous the condition might be termed simple hypertrophy, when their number is excessive it might be described as glandular hypertrophy of the endometrium. The retention and accumulation of secretion in the glands give rise to what is called cystic hypertrophy.

All local hyperplastic growths that project beyond the surface readily assume the form of **polypi**, sessile or pedunculated. Their shape varies with the manner in which they are pressed on by the uterine walls, but they are usually flattened. The elongated pedicle is due to the traction exerted on the polypus by the uterine contractions, set up by the presence either of the tumour itself or of blood and secretions collected behind it. Polypi, and particularly those which originate from the cervix, often make their appearance at the os uteri, and project through it into the vagina. Those that grow from the vaginal portion, whether sessile or pedunculated, sometimes appear at the vulva.

Uterine polypi are usually small, varying in size from that of a bean to that of a hazel-nut, though some are as large as a hen's egg. They nearly always enclose small cysts, especially those that grow from the cervix and the vaginal portion. Their surface is smooth, but is often pitted with small depressions and clefts resembling the crypts of the tonsils, which represent the mouths of glands and ruptured cysts. They are occasionally studded in places with papillary outgrowths. In the upper portion of the cervix, or in the body of the uterus, their surface is usually invested with columnar epithelium, although over some parts the epithelium is squamous. Polypi that spring from the lower portion of the cervical canal may be covered with squamous or with columnar epithelium, or with both kinds together.

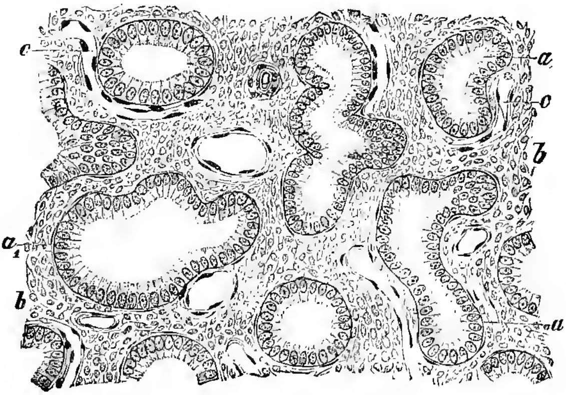

FIG. 528. HYPERPLASIA OF THE UTERINE MUCOUS MEMBRANE.

(*Section of a fragment of tissue removed from the uterus with the curette: hardened in alcohol:* × 150)

a a_1 transverse sections of the glands
b fibrous tissue of the endometrium
c blood-vessels

These hyperplastic growths of the mucous membrane are non-malignant, inasmuch as they do not destroy the underlying tissue. Their only practical importance lies in the fact that they frequently cause discomfort and leucorrhoea, and that when they are highly vascular (angiomatous polypi) they are apt to give rise to menorrhagia and metrorrhagia. It is also worthy of note that some of them tend to pass into sarcoma or carcinoma and so to become malignant (Art. 527).

References on Hyperplastic Growths of the Endometrium (see also Art. 323).

ACKERMANN: Glandular polypi of the anterior lip of the os uteri *V. A.* 43 **1868**
CORNET: *Polypes muqueux de l'utérus* Paris **1889**
HEINRICIUS: Chronic hyperplastic endometritis *A. f. Gynäk.* XXVIII **1886**
SCHATZ: Diffuse cystic fibro-adenoma *A. f. Gynäk.* XXII **1884**
SCHROEDER: *Z. f. Geburtsh.* II **1877**
STRATZ: Amyloid polypi *Z. f. Geburtsh.* XVII **1889**
ZAHN: Tuberculous polypi *V. A.* 115 **1889**

325. Inflammation of the muscular tissue of the uterus is known as **metritis**, and is characterised by swelling and cellular infiltration of its substance (Fig. 529 *a*); it is generally consecutive to endometritis, or induced by traumatic injury to the uterine wall. Severe forms lead to local suppuration and destruction of the muscular tissue, with partial fibrous or cicatricial induration. As an independent affection it is by no means common.

Atrophy of the wall of the uterus, during the period of its functional activity, is generally met with *post partum*, the muscular tissue undergoing fatty degeneration and resorption unaccompanied by adequate regeneration. This is most apt to happen after severe puerperal inflammation, which leads to partial destruction of the uterine parenchyma. Apart from the puerperal state atrophy may result from the traction exerted on the uterus by pedunculated uterine and ovarian tumours as they grow up out of the true pelvis, or by perimetritic adhesions, and from the lateral pressure of tumours in or adjacent to the uterine wall. In old age also the uterus usually becomes more or less atrophic, and as its muscular tissue disappears is reduced to a small and flabby thin-walled bag. The intima of the arteries is in general thickened, sometimes to such an extent that their channels are entirely obliterated and on section the cut ends stand out stiffly from the lax and wasted uterine tissue.

Hypertrophy of the uterus (uterine infarction), in which the body of the organ is more or less enlarged, supervenes *post partum*, and also independently of parturition: it depends in part on overgrowth of the muscular substance and in part on fibrous hyperplasia.

In many instances the enlargement is demonstrably associated with inflammatory conditions, and clinical experience also shows that at least some cases of uterine hypertrophy are directly consequent on metritis.

Uterine enlargements following parturition are occasionally due simply to increase of muscular tissue, no inflammatory changes being subsequently discoverable: the condition is therefore one of muscular hypertrophy, resulting from imperfect involution on the one hand, or on the other from excessive reproduction in replacement of the degenerate elements. In some cases the uterus after delivery passes into a state of inflammation, which is recognisable by its clinical symptoms, and when it subsides often leaves its traces in the form of perimetritic adhesions. When the uterus is examined *post mortem* before the inflammatory process has come to an end, its bulk is seen to be enlarged and its tissue beset with numerous patches of cellular infiltration (Fig. 529), aggregated mainly about the parts where the muscular wall is traversed by fibrous bands (*a*) and large blood-vessels. The uterine hypertrophy that results in such conditions is thus partly muscular and partly fibrous.

The new fibrous tissue is produced chiefly about the larger blood-vessels and surrounds the separate muscular fasciculi with layers of considerable thickness. Sometimes the intrafascicular fibrous tissue is also broadened. By the time the process is complete, the fibrous tissue has become dense and non-cellular, and the uterine wall is rendered hard and tough.

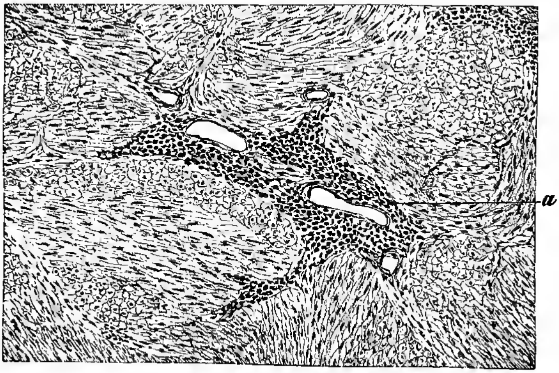

FIG. 529. METRITIS.

(*Section from a uterus four weeks after delivery: preparation hardened in Müller's fluid, and stained with alum-carmine:* × 50)

a patch of cellular infiltration in the intermuscular fibrous tissue

Similar conditions are often set up in the uterus by chronic endometritis, by frequent irritation of the vaginal portion from surgical applications, excessive coition, and so on, by dysmenorrhoea, and by chronic venous engorgement resulting from acute flexion, prolapse, habitual constipation, and uncompensated heart-disease of a serious kind. In these cases it is difficult to decide on histological grounds alone as to the respective shares in producing the fibrous and muscular hypertrophy which should be assigned to inflammation, to mere disorder of the circulation, and to uterine contraction induced by antecedent morbid conditions. The intermittent uterine swelling, pain, and tenderness that are usually observed would indicate that inflammation is in progress, though when the uterus comes under post-mortem examination it is rarely possible to detect any signs of inflammatory infiltration in its tissue.

Hypertrophy of the cervix sometimes supervenes without any obvious cause, sometimes is associated with displacements of the uterus and vagina that cause traction on the cervix or disturbance of its circulation, and sometimes follows upon chronic inflammation. The hypertrophy is usually limited to particular parts.

Hypertrophy of the vaginal portion is either general or limited to the lips of the external os. In the former case the vaginal portion retains its shape, but increases in length until in some instances it is visible at the vulva. When only one lip of the os is enlarged its form is usually distorted. The cause of this variety of hypertrophy is unknown. Hypertrophy consecutive to chronic inflammation and to parturition as a rule gives rise to irregular soft or hard enlargement of the external os.

The commonest cause of hypertrophy of the supravaginal portion of the cervix is primary prolapse of the vagina, the latter exerting a constant traction on the cervix, at least in cases where the uterus is fixed in its normal position and does not prolapse with the vagina. In other cases the cause cannot be determined. In both varieties the vaginal fornix is displaced downwards, and when the cervical hypertrophy is great may even become completely inverted. The bladder, the recto-uterine pouch of peritoneum, and sometimes the vesico-uterine pouch also, are dragged downwards, but the fundus of the uterus remains at its normal level.

Hypertrophy of the middle portion of the cervix is usually due to prolapse of the anterior wall of the vagina, which causes the anterior lip of the os to be dragged upon, especially if the uterus is fixed by morbid adhesions or by a tumour, and so cannot yield to the traction. The enlarged middle portion above the anterior lip pushes down the anterior fornix, and as this carries with it the posterior vesical wall a diverticulum of the bladder is produced. The enlarged posterior lip, on the other hand, is intravaginal, as it lies below the line of attachment of the fornix. The hypertrophied cervix, as in the case of uterine prolapse, occasionally protrudes from the vagina. The anterior fornix is then effaced, but the posterior fornix retains its normal situation or is at most a little depressed (SCHROEDER).

The substance of the hypertrophied cervix is made up of fibrous tissue and muscular fibres; but the latter are often disproportionately scanty, the enlargement being thus in many cases due essentially to fibrous overgrowth.

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DITTRICH: The muscular tissue of the puerperal uterus under morbid conditions *Prager Z. f. Heilk.* x 1889
FISCHEL: Abscess of th[illegible]. 1886

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SÄNGER: Involution of the muscular tissue of the puerperal uterus *Beiträge z. path. Anat. und klin. Med.* (*Wagner's Festschrift*) Leipzig **1887**
SCANZONI: *Die chronische Metritis* Vienna **1863**
SCHROEDER: *Handbuch der Krankh. der weibl. Geschlechtsorgane* **1887**
DE SINÉTY: *Manuel de gynécologie* Paris **1879**

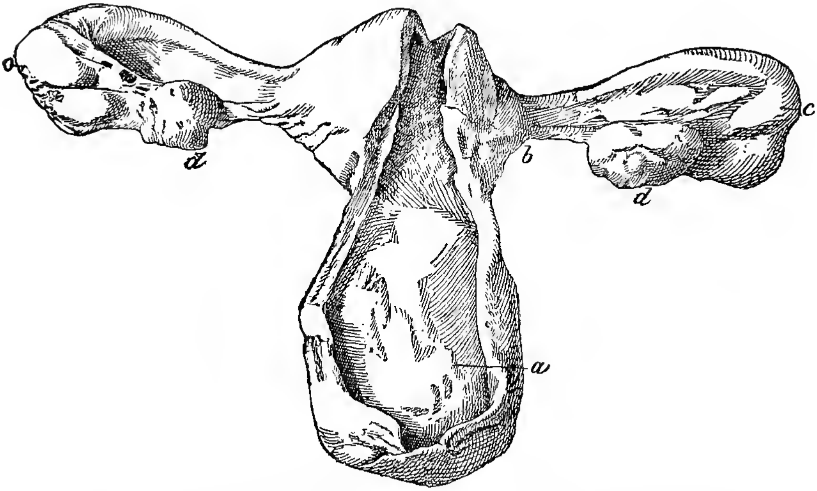

FIG. 530. HAEMATOCOLPOS, HAEMATOMETRA, AND HAEMATOSALPINX.

(*From a case of acquired occlusion of the vagina and of the abdominal ostia of the tubes: one-half the natural size*)

a vagina dilated into a sac
b dilated uterus
c dilated tubes, adherent to the ovaries *d*

326. **Stenosis** and **atresia** of the genital canal are either dependent on malformation (Art. 319), or the result of inflammatory swelling, of tumours, or of cicatricial contractions and adhesions. Occlusion of the internal os takes place generally in old age, and is due to inflammation, or in some cases to uterine flexion. Occlusion or constriction of the external os is caused by cauterisation, or by laceration during parturition. In the vagina (Fig. 530) stricture follows upon injuries inflicted during parturition or violation, ulceration and gangrene, cauterisation, and so on.

Such constrictions give rise to no ill effects so long as no menstrual or other discharge seeks to escape from the uterus; but as soon as menstruation begins dysmenorrhoeal distress sets in, and blood collects behind the point of obstruction. The initial seat of the accumulation naturally depends on the position of the barrier. In imperforate hymen the vagina (Fig. 530 *a*) is first distended, and then the uterus (*b*): in atresia of the external os (Fig. 518) the uterus alone is distended (*c*). In the former case a **haematocolpos** and afterwards a **haematometra** is produced, in the latter case a haematometra only. The retained blood assumes a chocolate-brown or brownish-black tint, and is liable to become inspissated.

The distended cavities sometimes reach a considerable size. Not infrequently the ever-accumulating blood passes into and dilates the tubes also (Fig. 530 *c*), causing **haematosalpinx**: if any of the retained blood escapes from the tube into the abdominal cavity, it is apt to set up adhesive inflammation in the peritoneum.

When the occlusion of the external or internal os does not take place until after the menopause, the liquid that gathers in the uterus is serous or mucous, and the condition is termed **hydrometra**. In very rare instances, in which both the external and the internal os are occluded, the uterus and cervix are distended independently, and the organ assumes an hour-glass shape.

Purulent catarrh supervening behind the point of obstruction causes purulent liquid to gather in the uterus, distending it into a **pyometra**. In uterine tuberculosis the contents of the pyometra consist of cheesy pus (Fig. 527 *b*), and in broken-down cancer of the body of the uterus the retained matter is turbid and white or blood-stained.

As the blood continues to gather in the haematometra, the obstructing tissue sometimes gives way under the increasing pressure, having perhaps been previously softened by gangrenous inflammation. In other cases the uterus or vagina ruptures, and its contents escape into the surrounding parts: in very rare cases secondary rupture and evacuation take place subsequently into the bladder or vagina. A tube that has been blocked by previous inflammatory adhesion is liable to rupture in like manner (Fig. 518 *e*), and so to empty its contents into the abdominal cavity.

A hydrometra rarely grows to the size attained by a haematometra, and after a time it ceases to enlarge, so that rupture into the adjacent parts is uncommon. When the occlusion of the cervix is incomplete or yielding, the liquid from time to time escapes into the vagina, and then proceeds to collect afresh.

In exceptional cases the contents undergo decomposition and evolve gas: to this condition the term **physometra** is applied.

When the genital canal is duplicated in whole or in part, and one side of it is occluded below, a unilateral haematometra (Fig. 518 *c*) or haematocolpos may be produced.

References on Occlusion of the Utero-vaginal Canal.

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Dohrn: Atresia vaginae *A. f. Gynäk.* X **1876**

Eppinger: *Prager Vierteljahrsschr.* **1873**

Freund: Morbid anatomy of duplication of the genital canal *Z. f. Geburtsh.* I **1877**

Graefe: Ten cases of vaginal cysts *Z. f. Geburtsh.* VIII **1882**

Graf: *V. A.* 19 **1860**

Greulich: *Art.* Haematometra *Eulenburg's Realencyklop.*

Hennig: *Z. f. Med. Chir. u. Geburtsh.* V **1866**

Klob: *Pathol. Anat. der weiblichen Sexualorgane* Vienna **1864**

Puech: *De l'atrésie des voies génitales de la femme* Paris **1865**

Rheinstädter: Occlusion of the cervix from cauterisation *A. f. Gynäk.* XIV **1879**

Staude: *Z. f. Gebh. u. Frauenkrankh.* Berlin **1876**

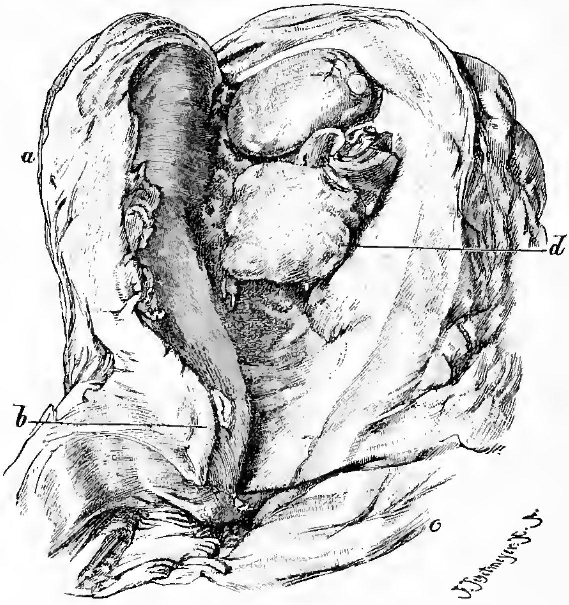

Fig. 531. Fungous carcinoma of the endometrium.

(*The growth is situated on the posterior wall of the body of the uterus: two-thirds of the natural size*)

a body of the uterus *b* cervix *c* vagina *d* tumour

327. Of the **tumours** of the uterine mucous membrane **carcinoma** is the commonest and the most important. It usually makes its appearance in the vaginal portion and in the cervix (Fig. 532 *b*), less frequently in the body of the uterus (Fig. 531 *d*). It may start in a previously healthy mucous membrane

or in a part which has undergone hyperplastic overgrowth, and assumes the form of projecting or intramural nodes or of papillomatous excrescences.

At the start the growth is local, but it very soon extends over the circumjacent endometrium and underlying portions of the uterine tissue.

Carcinoma starting from one of the lips of the external os tends to invade the contiguous portions of the vagina, and at the same time to penetrate deeply into the vaginal portion of the cervix. Growths of the cervix itself extend round the whole circumference of the cervical mucous membrane, and infiltrate the underlying muscular tissue. Carcinoma of the body produces nodes in the wall at the fundus or about the middle zone (Fig. 531 *d*), or occasionally gives rise to a ring of excrescences encircling the inner surface of the uterus and penetrating more or less deeply into its muscular substance. Now and again the growth spreads laterally over the entire inner surface of the uterine cavity.

On section the tissue of a uterine carcinoma usually appears whitish, opaque, and easily distinguishable from the redder and somewhat translucent uterine tissue. Sooner or later the prominent parts of the growth undergo softening and disintegration, and thus form a carcinomatous ulcer

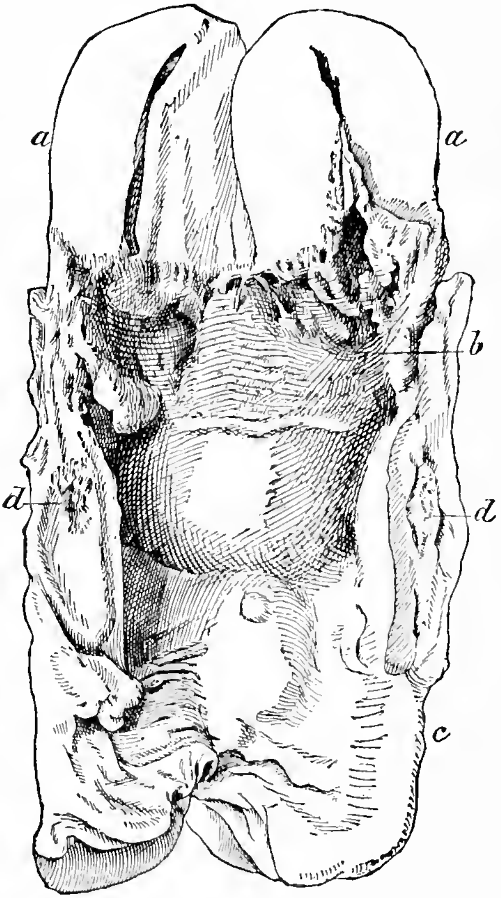

FIG. 532. CARCINOMA OF THE VAGINAL PORTION OF THE UTERUS AND OF THE VAGINA.

(*Longitudinal section: three-fifths of the natural size*)

a body of the uterus
b carcinomatous ulcer
c lower part of the vagina
d infiltrated wall of the vagina

(Fig. 532 *b*), whose ragged uneven surface is covered with necrotic tissue and often with extravasated blood. As the disease extends new portions of the uterus, and it may be the adjacent parts of the vagina, are invaded by the epithelial infiltration, and are then destroyed by ulceration.

The pelvic connective tissue and peritoneum, the bladder, and the rectum are in like manner involved, and wherever the carcinoma extends neoplastic proliferation and induration are induced. Contiguous structures become adherent, and their tissue is beset with carcinomatous nodes. The lymph-glands of the pelvis also swell up, and undergo carcinomatous infiltration.

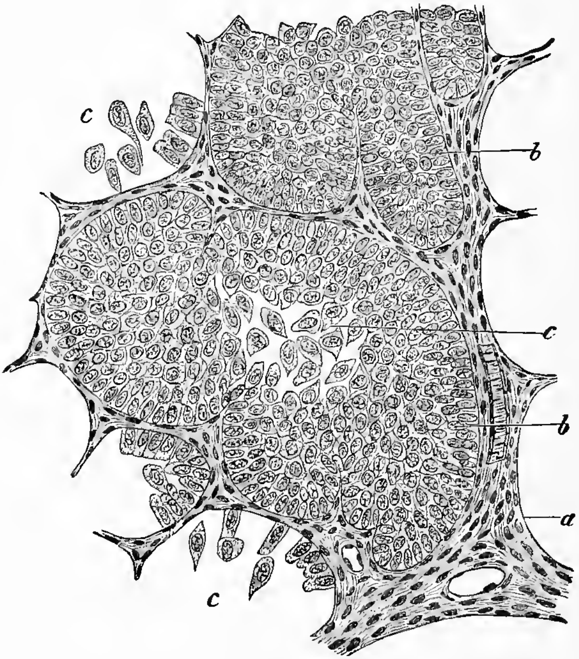

FIG. 533. ADENOCARCINOMA OF THE BODY OF THE UTERUS.

(*Section of a piece of tissue removed from the uterus with the curette: preparation hardened in alcohol, and stained with haematoxylin* : × 200)

a cancer-stroma *b* nests of cancer-cells *c* isolated cancer-cells

It thus comes about that in the course of time large portions of the uterus and usually of the vagina also are destroyed, and the place of the cervix, the vaginal portion, and the upper part of the vagina, is occupied by a large cavity (Fig. 532 *b*), whose eroded and infiltrated walls are covered with foetid and decomposing shreds and detritus. When the neoplastic infiltration extends backwards the wall of the rectum is often cancerous and broken through at several places: when it extends forwards, a passage is opened into the bladder (vesico-vaginal or vesico-uterine fistula). Even when the process has started in the cervix, the fundus of the uterus is in many cases the only portion of the organ that remains.

Carcinoma of the uterus is in some instances glandular or adenomatous, and starts from the glandular epithelium, in other cases epitheliomatous, proceeding from the squamous epithelium.

Adenocarcinoma is met with chiefly in the body of the uterus, and begins with an excessive and atypical hyperplasia of the glandular epithelium (Fig. 533 *b*), or with the formation of papillary excrescences in the interior of the glands. When fully developed the growth exhibits the characters partly of a columnar-celled adenocarcinoma and partly of an ordinary soft or hard (medullary or scirrhous) cancer. Squamous-celled epithelioma may however develope from the glands of the cervix, the glandular columnar cells producing new squamous epithelium (Fig. 534 *e f*). Cancers that start from the squamous epithelium of the vaginal portion are of the epitheliomatous or squamous-celled type.

Uterine carcinoma is commonest between the ages of 30 and 50; but women who are under 30 or over 50 are not exempt from it.

Sarcoma of the uterine mucous membrane takes the form of soft nodose or papillomatous (botryoidal) vascular growths, which penetrate into the muscular wall. They are made up of spindle-cells, round cells, or polymorphous cells, and occasionally include giant-cells also (myeloid sarcoma). There is moreover a peculiar variety, described as deciduo-cellular sarcoma, the cells of which resemble those of the decidua, and in most cases appear actually to proceed from the persistent decidual tissue of a recent pregnancy (Art. 334).

The so-called **fibroid** is the commonest tumour of the muscular wall of the uterus; it occurs in the form of sharply-defined rounded growths varying from the size of a pea to that of a full-time pregnancy (Fig. 522 and Fig. 527 *d*). The growths consist of muscular tissue chiefly (leiomyoma), of muscular and fibrous tissue (fibromyoma), or of fibrous tissue entirely (fibroma). The muscular tissue exhibits a reddish-white, the fibrous tissue a whitish colour.

Fibroids are usually developed in or after middle age, though they are not infrequent in young women. They may be single or multiple. Most of them are poorly supplied with vessels, though in large tumours there are often parts that are highly vascular or even angiomatous. Some enclose wide smooth-walled spaces filled with clear lymph.

In the substance of the tumour signs of degeneration (chiefly those of fatty and waxy change) are often apparent, whereby the muscular tissue is more or less completely destroyed, and what was originally a fibromyoma is converted into a pure fibroma. This change is especially apt to occur during the puerperal period, the fibromyoma participating in the general involution of the uterine wall. Calcareous infiltration sometimes follows upon fatty and necrotic changes in the tumour, and is occasionally so abundant as to cause its petrifaction.

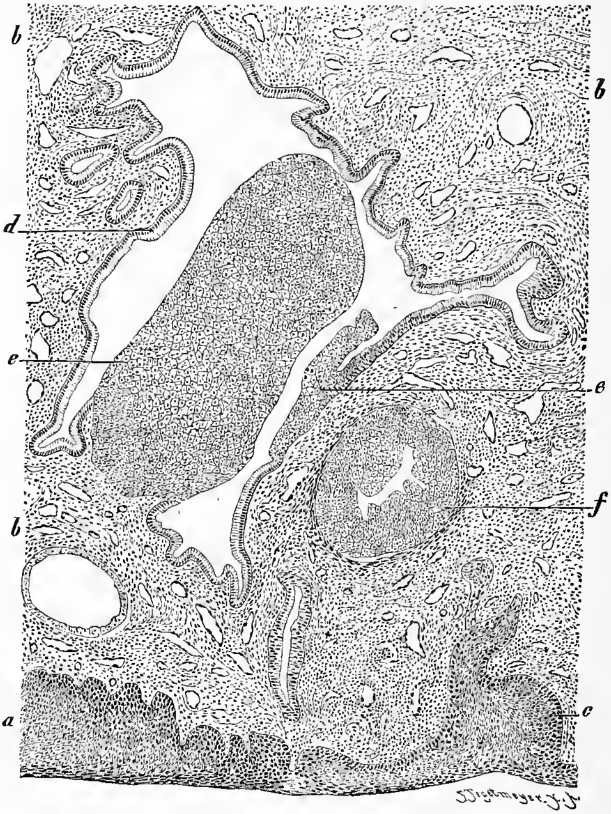

FIG. 534. INITIAL STAGE OF UTERINE CARCINOMA.

(*Section from the vaginal portion of the uterus: hardened in alcohol, and stained with Bismarck-brown:* × 45)

a	surface epithelium
b	fibrous tissue
c	surface epithelium growing into the underlying tissue
d	dilated gland
e	processes of glandular epithelium growing into the gland *d*
f	transverse section of a gland whose columnar epithelium has been transformed into stratified squamous epithelium

The fibrous tissue of a fibroid may be either hard and coarse, grating under the knife, or soft and flabby, in which case it is usually sodden and oedematous. Sometimes it is partly transformed into mucoid tissue, or its constituents undergo softening and liquefaction, giving rise to cysts containing a liquid that is clear or turbid from admixture with fatty cells. Occasionally the greater part of the tumour is thus destroyed.

Inflammation is liable to be set up in a fibroid subjected to operative procedures, and may end in suppuration or gangrene of its substance.

In certain cases fibroids show in places a tendency to excessive cellular proliferation, which may cause them to assume a sarcomatous character.

Fibroids are usually seated in the body of the uterus, more rarely in the cervix; according to their position they are distinguished as submucous, interstitial, or subserous. Subserous tumours soon project above the external surface of the uterus, and may even become pedunculated. Subsequent cystic excavation sometimes gives them the appearance of ovarian cysts. Submucous fibroids grow into the cavity of the uterus, and when they become pedunculated are described as fibrous or fibro-muscular polypi. Interstitial fibroids raise both the internal and the external surface, and have a tendency to protrude from one or other as they grow larger.

Fibroids often cause the uterine wall to become hypertrophic; but when they are numerous and compress the uterine tissue between them it is apt to undergo local atrophy.

The growth of a fibroid is slow, and many of them never attain to any considerable size.

Submucous and interstitial fibroids usually give rise to leucorrhoea and metrorrhagia. Some are spontaneously extruded, the uterine contractions forcing them downwards, while the overlying mucous membrane becomes attenuated and eroded, and their attachments to the wall of the uterus are gradually loosened. During the process of spontaneous extrusion the tumour not infrequently sloughs *en masse* and gives rise to metritis or endometritis.

Sarcoma of the uterine wall generally takes the form of nodose tumours occupying the same positions as fibroids: they are occasionally multiple. It is probable that in most cases they are produced from fibroids by a kind of metaplasia. Their tissue is in some cases made up chiefly of round cells, in others chiefly of spindle-cells: portions of them may possess a myxomatous texture. They are not common.

Lipoma is very rarely met with in the uterus.

Tumours of the uterine wall now and then enclose cysts and glandular tubules lined with epithelium: these are probably remnants of the wolffian body or paroöphoron.

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BISCH: *Du cancer prim. du corps de l'utérus* Paris **1892**
BREUS: *Ueber epithelführende Cystenbildung in Uterusmyomen* Vienna **1894**
EDIS: Myoma *Brit. Gynæc. Journ.* 1 **1885**

Gusserow: Tumours of the uterus *Billroth's Handb. d. Frauenkrankh.* II Stuttgart **1886**
Haeckel: Melanotic tumours *A. f. Gynäk.* XXXII **1888**
Hauser: Glandular tubules in uterine myoma *Münch. med. Woch.* **1893**
Hofmeier: Carcinoma of the body of the uterus *Z. f. Geburtsh.* XXXII **1895**
von Kahlden: Sarcomata of the uterus *Ziegler's Beiträge* XIV **1893** (with references)
Klein: Deciduo-cellular giant-celled sacroma *A. f. Gynäk.* XLVII **1894**
Kürsteiner: Papillomatous cancer *V. A.* 130 **1892**
Länderer: Adenocarcinoma corporis uteri *Z. f. Geburtsh.* XXV **1892**
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CHAPTER CVI

THE VAGINA

328. The **vagina** has the form of a canal flattened from before backwards, whose walls are composed of coarse fibrous tissue interspersed with fasciculi of non-striated muscular fibres.

The inner surface is lined with mucous membrane beset on the anterior and posterior walls with warty prominences and transverse ridges (*columnae rugarum*), which are well marked in the virgin, and tend to disappear after repeated parturitions. The papillae are covered over with stratified squamous epithelium, and the membrane is usually destitute of glands; though deep crypt-like depressions exist between the papillae and rugae which bear some resemblance to glands (EPPINGER). Small aggregations of lymphadenoid tissue are interspersed throughout the submucous connective tissue, but their number varies greatly in different women.

The commonest morbid affection of the vagina is inflammation, known as **vaginitis** or **colpitis**; it assumes the form of desquamative or purulent catarrh, or of diphtheritic sloughing. Vaginal catarrh is generally due to gonorrhoeal infection, but it is worthy of note that as a rule the *Gonococci* multiply in the mucous membrane of the cervix uteri and urethra only, the vaginal secretion itself being free from them. Among other causes giving rise to vaginal inflammation are irritant and corrosive drugs, injections, operative interference, dirty pessaries, and the like. Vaginitis also occurs in connexion with measles, scarlet fever, small-pox, cholera, etc.

In recent cases of acute vaginitis the mucous membrane is reddened, lax, and soft, the rugae being greatly swollen. The secretion contains shed epithelium and pus-corpuscles. In rare cases inflammatory vesicles are formed in the surface epithelium (*vaginitis vesiculosa*). In chronic vaginitis, such as follows on an acute attack or is gradually induced by constantly-repeated irritation of the vagina, arising it may be from frequent coition or from the friction of pessaries and other foreign bodies, the secretion is whitish and cream-like or yellow and puriform. The mucous membrane is in some cases swollen and reddened, or even studded with ecchymoses; in others it is smooth, tough, and rigid, of a reddish-brown or slaty tint; and it may be spotted with grey patches. The tissue is infiltrated with leucocytes, and occasionally (Eppinger, Ruge, Chiari) encloses circumscribed cellular nodes like little follicles, which lie beneath the papillary stratum, and raise the surface into granular-looking prominences (granular or follicular vaginitis).

When the condition of irritation is long maintained, as in cases of chronic gonorrhoea of the uterus and urethra, the papillae of the vaginal mucous membrane are apt to become enlarged and the connective tissue thickened, the result being a condition of diffuse or circumscribed warty overgrowth of the membrane. When the overgrown papillae are large and numerous they encroach considerably on the lumen of the canal. In old women infiltrated portions of the vaginal wall occasionally lose their epithelium, and when these come into contact they are liable to grow together and cause obstruction or partial obliteration of the canal (*colpitis vetularum*).

Erosions, necroses, and ulcers are produced by decomposing secretions and by the persistent pressure of foreign bodies introduced into the vagina. Round ulcers like those of the stomach have been described by Zahn. Suppuration also occasionally makes its appearance, and in rare cases spreads like a phlegmonous inflammation beneath the mucous membrane, causing wide-spread destruction of the vaginal tissue. Foreign bodies, such as pessaries, that have lain for a long while in the vagina are liable to become encrusted with calcareous salts, or so overgrown with granulation-tissue that they can be removed only after free incision.

Diphtheritic vaginitis, if we exclude puerperal cases, is usually due to gangrenous necrosis supervening in uterine cancers and polypi, vesico-vaginal and recto-vaginal fistulae, irritation from pessaries, or general infections such as measles, small-pox, scarlet fever, typhoid fever, cholera, etc. The diphtheritic sloughing is at times limited to a few spots in the vagina, in others it extends over the greater part of its wall. The mucous membrane is intensely swollen, and overlaid with white, grey, or greenish sloughs and false-membranes. When only the most superficial layers are involved the false-membranes may be of a croupous or fibrinous character. Wide-spread sloughing naturally implies repair by cicatrisation, and this ends in contraction and even at times in partial obliteration of the canal. Ulcers produced on apposed surfaces, whether by diphtheritic necrosis or otherwise, are liable to grow together as they heal by granulation, and so to bring about complete occlusion of the vagina. In other cases the canal is crossed by fibrous bands and membranes. In very old women the vaginal fornices tend to disappear altogether.

Vaginal **cysts**, single or less frequently multiple, are sometimes met with in the walls of the canal. Their liquid contents are as a rule clear and serous, but occasionally they are stained red or brown by admixture with blood-pigment. Their origin is probably different in different cases; some are due to the accumulation of secretion within the above-mentioned crypts of the mucous membrane, others perhaps arise by the distension of persistent remnants of the wolffian ducts (Veit) or from diverticula of the ducts of Müller (Freund). Preuschen and Hückel have pointed out that in some women the vaginal walls are provided with true glands, and that these may be transformed into cysts. In other cases again the cysts appear to be produced by liquid accumulating in the lymph-spaces and lymphatics of the vaginal wall (Klebs).

In the vaginas of pregnant and recently-delivered women, and even of women who have not been pregnant, small air-containing blebs have frequently been observed; they vary from the size of a millet-seed to that of a hazel-nut. These vesicles are commonly known as gaseous cysts or **air-cysts** (Schroeder): other authorities refer to them as *aërocystides vaginae* (Chiari), cystic colpohyperplasia (Winckel), emphysematous vaginitis (Zweifel), or vaginal emphysema (Eppinger).

Winckel affirms that they are caused by the enclosure of putrefactive gases between coherent folds of the mucous membrane; Eppinger, Schmolling, Chénevière, and Ruge believe that air enters and distends into cysts the meshes of the vaginal connective tissue; Zweifel and Hückel regard them as dilated glands; Schroeder and Näcke as excavated follicles; and Klebs as lymphangiectases. According to Chiari, they are developed from the lymphatics, and enclose proliferous endothelial cells, some of which are converted into giant-cells. The gas is probably air that has penetrated from without.

Tuberculosis of the vagina is secondary either to uterine tuberculosis or in very rare instances to tuberculous disease of the vulva.

The commonest **connective-tissue tumours** met with in the vagina are fibroma, fibromyoma, myxoma, and sarcoma, the former two being much less frequent than the like growths of the uterus. Fibroma, myxoma, and sarcoma occur in the form of multiple polypi scattered over the entire canal. Rudnewa has recorded a case of vaginal rhabdomyoma.

Primary carcinoma is met with in the vagina both as a circumscribed growth and as a diffuse nodose infiltration of the greater portion of its wall. As the growth breaks down it gives rise to cancerous ulcers of various sizes.

Certain **epizoa** and **epiphytes** find lodgement in the vagina, *Trichomonas vaginalis*, *Oxyuris vermicularis*, *Oïdium albicans*, *Leptothrix vaginalis*, and the various kinds of bacteria, being the most important. *Oxyurides* (thread-worms) occasionally gain entrance from the rectum, and give rise to some irritation and itching. *Oïdium* (thrush-fungus) forms whitish films on the mucous surface: it is met with chiefly in lying-in women. The inflammation set up by such vegetable parasites has been termed mycotic vaginitis (VON HERFF).

When the vagina is unduly lax and overstretched, either from chronic inflammation or from frequent child-bearing, the anterior or the posterior wall, or both together, are liable to prolapse into the canal or even through the vulva. Such **prolapse** is facilitated by any loosening of the attachments of the vagina to its surroundings. Should the anterior wall of the rectum or the posterior wall of the bladder be also relaxed, they yield to the traction of the falling vagina and prolapse with it: the rectal and vesical diverticula so produced are termed respectively **rectocele** and **cystocele**. On the other hand, dilatation of the bladder or of the rectum sometimes leads to prolapse of the vagina. In rare cases the posterior wall of the vagina is pushed down by an ovarian tumour (vaginal ovariocele), by coils of intestine (vaginal enterocele), or by liquid collecting in and deepening Douglas's pouch. Partial prolapse of the uterus is often accompanied by prolapse of the lower part and inversion of the upper part of the vagina. In other cases the vaginal prolapse is the primary condition, the subsequent descent of the uterus being due to the traction exerted on it by the vaginal wall. In total prolapse of the uterus the entire vagina is turned inside out.

Laceration and partial destruction of the vaginal tissues are not infrequently brought about by the stretching and bruising to which they are subjected during parturition. The most important traumatic lesions of the kind are those that give rise to the formation of fistulous openings between the genital canal and the bladder or rectum. Under the persistent compression of the soft structures against the superior posterior edge of the os pubis by the presenting part of the child, the vaginal wall is bruised, becomes necrotic, and ultimately gives way. The commonest result is a **vesico-vaginal fistula**: fistulae between the urethra or one of the ureters and the vagina, and between the bladder and the cervix, are much rarer. The fistulous opening may be narrow or wide, the edges thin and sharp, or callous and thickened. Apart from those due to traumatic injury in child-birth, vesico-vaginal and recto-vaginal fistulae are oftenest produced by the ulceration set up round a pessary, and by cancerous erosion of the vagina, rectum, and bladder.

The larger **lacerations** produced during child-birth usually involve the posterior vaginal wall and perineum, and sometimes extend to the rectum. Extensive lacerations do not heal up spontaneously, but require a plastic operation for their repair. Sometimes the perineal rupture alone unites, the rent in the upper portion of the septum between rectum and vagina remaining as a **recto-vaginal fistula**.

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CHAPTER CVII

THE PELVIC PERITONEUM AND CONNECTIVE TISSUE

329. **Inflammations** of the pelvic peritoneum (perimetritis), and of the loose cellular tissue underlying it (parametritis), are of somewhat frequent occurrence.

Parametritis, or inflammation of the subserous connective tissue about the vagina, broad ligaments, and iliac fossae, is in the majority of cases a puerperal affection; but it is also liable to follow operations on the vagina and cervix, and is occasionally associated with acute inflammations of the genital mucosa, ulcerations of the rectum, pelvic ostitis, etc. In its early stages it is indicated by the effusion of serous, cellular, and sometimes haemorrhagic exudations into the tissue; in cases of pyogenic infection it often assumes a phlegmonous character, and sooner or later issues in general suppuration, with the formation of abscesses that tend to burrow and break into one or other of the adjoining cavities.

The exudations are usually re-absorbed, particularly in the puerperal and traumatic cases, and if the process has continued for some time a certain amount of induration of the affected tissues takes place. It must however be borne in mind that parametritic collections of pus (residual abscesses) sometimes remain quiescent for years without losing their power of starting fresh inflammation, and even of inducing pyaemic metastases in other organs.

Perimetritis, or pelvic peritonitis, follows upon inflammation of the uterus, tubes, or ovaries, and upon parametritis; it is a common affection both in the puerperal state and independently of parturition.

The inflammation usually spreads directly from the above-named parts to the peritoneum. It is sometimes set up by effusions of blood from the ovaries and tubes which collect in the lower parts of the vesico-uterine and recto-uterine pouches, especially when the blood coagulates into solid masses or deposits fibrin on the serous surfaces. Perimetritis is also not infrequently but part of a general peritonitis, or starts from inflammations in other structures within or adjacent to the pelvis, such as the bladder, the vermiform appendage, the rectum, and the pelvic bones.

The inflammation frequently assumes a purulent or putrid character, especially in puerperal cases, and then usually terminates in fatal general peritonitis. Fibrinous, sero-fibrinous, or fibrino-purulent perimetritis leads as a rule to the formation of adhesions and cicatricial bands uniting and agglutinating the various organs of the pelvis. The position of these adhesions depends upon the original starting-point of the inflammation. The posterior wall of the uterus is peculiarly liable to become adherent to the anterior wall of the rectum, to the ovaries, and to the fimbriae of the tubes (Figs. 504 and 520). In other cases the uterus is adherent to the bladder, intestines, omentum, etc. The adhesive membranes are sometimes so numerous and dense that the tubes and ovaries are inseparably matted together. In recent cases the interstices of the bands contain inflammatory exudations, and in later stages they often enclose collections of clear serous liquid, which have somewhat the appearance of cysts. Liquid or clotted blood is occasionally found interspersed among the adhesions: it is either derived from the fragile vessels contained in the membranes themselves, which are highly vascular, or is due to effusions from the peritoneum, tubes, or ovaries. A circumscribed collection of blood thus encapsuled is called a **pelvic haematocele**, retro-uterine or ante-uterine, according to its position. Retro-uterine haematocele is the more common, and may attain such dimensions as to push the uterus forward, and even to overlap it. Ante-uterine haematocele depresses the uterus downwards into Douglas's pouch. Each fresh haemorrhage, producing new clots, gives rise to fresh irritation and rekindles the inflammation, which persists until the extravasation is either re-absorbed or completely encysted.

When the adhesions enclose collections of pus, these like parametritic abscesses are liable to break through into the neighbouring hollow viscera.

In some rare cases primary **connective-tissue tumours** develope in the broad and round ligaments of the uterus. Subserous pedunculated uterine fibroids growing between the two layers of the broad ligament are commoner; and ovarian tumours also occasionally extend into the ligament and push its serous surfaces apart. According to MARCHAND, small accessory suprarenals are sometimes intercalated in the broad ligaments near the ovaries. Uterine and vaginal cancers not infrequently break into the subserous connective tissue, and at times invade the peritoneum.

On and about the tubes are often found large numbers of small **cysts**, lined with columnar epithelium, which probably originate in persistent remnants of the wolffian body (paroöphoron). Large parovarian cysts, developed from the epoöphoron or organ of Rosenmüller, are met with in the broad ligament.

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CHAPTER CVIII

THE PUDENDA

330. The morbid changes of the **external genitals** in women correspond generally with those of the integument covering other portions of the body, though those of the vestibule are more akin to affections of the mucous membranes.

The various **inflammations** described in Section VIII under the heads of erythema, eczema, *herpes progenitalis*, prurigo, soft and hard chancre, *condyloma latum*, acne, furunculosis, phlegmon, gangrene, erysipelas, and lupus, are more or less common about the vulva.

One of the most frequent causes of inflammation of the mucous membrane is infection by *Gonococcus*. More or less intense inflammation is also excited by acrid catarrhal secretions from the internal genitals, by uncleanliness, by friction in walking, etc. The inflammation is manifested by redness and swelling, and often by the secretion of pus from the mucous surfaces, while excoriations are often produced about the labia.

Diphtheritic and croupous inflammations arise in connexion with diphtheritic enteritis (dysentery), puerperal septicaemia, measles, typhoid fever, diphtheria, scarlet fever, cholera, etc.; in some cases they extend to the vulva by continuity from the inflamed intestine, vagina, or uterus. Such inflammations occasionally issue in gangrene.

Gangrene also results from specific phagedaenic ulceration, grave disorder of the circulation, injuries during child-birth, venous thrombosis, haemorrhage, etc.

Noma of the vulva is analogous in its course and symptoms to noma of the cheek (*cancrum oris*).

A peculiar form of **atrophy** of the vulva and perineum, associated with cicatricial contraction of the integument, has been described by Breisky and Orthmann under the name of *kraurosis vulvae*. The affected tissue becomes dry and brittle, and its surface looks white and shiny. The normal folds are effaced, and the vulvar orifice is apt to become constricted. The cause of the affection is unknown.

Venous **engorgement** within the pelvis and in the lower limbs, due for example to pregnancy or to valvular disease of the heart, gives rise to distension of the vulvar veins; and the resulting

oedema sometimes causes the pudenda, and in particular the labia majora, to swell enormously. Inflammatory oedema is generally due to primary vulvitis, but it also appears in connexion with inflammations in adjacent parts, such as the pelvic bones or pelvic cellular tissue.

The hyperplastic and **fibromatous growths** of the external genitals are of special interest. They are by no means uncommon, some of them being congenital or at least having their roots in some congenital anomaly of texture, others resulting from recurrent or chronic inflammation or from persistent lymphatic and venous engorgement. In the first place the labia minora and the prepuce of the clitoris are liable to vary greatly in bulk, the former in old women sometimes projecting so far beyond the labia majora (Fig. 535 *b*) that they look like the foetal parts. In many African races the labia minora and the praeputium clitoridis are enormously developed, constituting what is known as the 'Hottentot apron.' A second variety of hyperplastic overgrowth assumes the form of circumscribed papillomatous excrescences, and of nodose or lobose polypi, which are usually soft, and spring from the labia or from the integument of the clitoris. In another group of cases particular portions of the external genitals undergo elephantoid enlargement, the labia majora for example sometimes attaining such enormous dimensions that they reach as far as the knees.

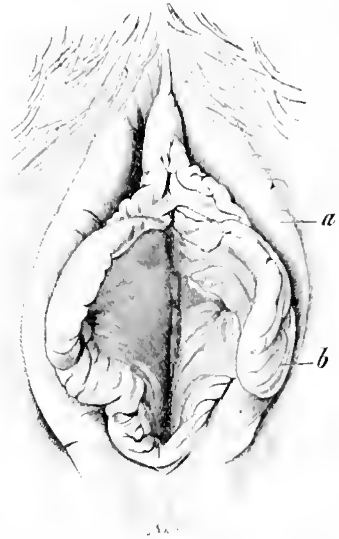

FIG. 535. HYPERTROPHY OF THE LABIA MINORA.

(*Seven-ninths of the natural size*)

a labia majora
b enlarged labia minora

Most of the papillomatous outgrowths are condylomata of inflammatory origin; less frequently they are of the nature of non-inflammatory warts.

The circumscribed nodose and polypous tumours are generally fibromatous, and consist of loose and often oedematous fibrous tissue; but some contain patches of mucoid tissue, and are then described as myxomata or myxofibromata.

Elephantiasis, affecting some considerable portion of the pudenda, produces enlargements that are firm and bacon-like, soft and fibrous, or sodden and gelatinous in consistence; the elephantoid parts are either uniformly enlarged or in various ways distorted and deformed. Some cases are non-congenital, and are due to inflammation and lymphatic engorgement, resulting from disease of the lymph-glands and the larger lymph-vessels: in other cases the overgrowth is simply hyperplastic, and is either congenital or dependent on some congenital tendency, such cases being included under the terms pachydermatocele, elephantoid molluscum, or elephantiasis mollis.

Elephantoid hyperplasia occasionally starts in a congenital **lymphangiectasis** or a cavernous cystic **lymphangioma** of the labia majora or of the inguinal region. The deeper portions of the tumour are at times converted into adipose tissue.

Apart from hyperplastic growths of this kind **carcinoma** is the commonest tumour of the pudenda; it originates either in the integument covering the clitoris or in the labia, and gives rise to papillomatous or nodose growths, or to corroding ulcers that sometimes spread over a wide extent of surface. **Sarcoma** and **lipoma** are uncommon: they usually start from the labia majora. Leiomyomata sometimes spring from the superficial attachments of the round ligaments.

Peritoneal **cysts** of the labia majora are produced by the dilatation of abstricted portions of the canal of Nuck. Other liquid-containing cysts of the vulva are due to the retention of glandular secretions or to lymphangiectasis.

Not infrequently comedones and atheromatous cysts originate from the sebaceous glands of the labia majora and minora. Dermoid cysts are of rare occurrence.

The outlets of the mucous **glands of Bartholin** lying immediately behind the *bulbus vestibuli*, which open on either side into the groove between the hymen and the labium minus, are sometimes occluded, and then the secretion accumulating within the ducts dilates them into one or more cysts. In some cases the cysts rupture into the vagina (VON RECKLINGHAUSEN), in others the cyst-wall becomes proliferous and is thereby thickened.

In purulent inflammation of the vestibule, such as that characteristic of gonorrhoea, the glands of Bartholin are liable to become inflamed, and pus accumulates in their ducts: the inflammation often persists in them long after it has subsided in the vagina and vestibule. A case of adenoma of Bartholin's glands has been described by COËN.

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CHAPTER CIX

THE PREGNANT UTERUS AND THE OVUM

331. When the fecundated ovum lies within the **uterus** and begins to develope, the **muscular wall** of the organ undergoes certain changes, which are manifested chiefly by the enlargement and multiplication of its constituent elements. The bulk of the uterus grows with the growth of the ovum, and its muscular fibres become more numerous and increase in size until they are eight or ten times the ordinary length. The muscular hypertrophy is accompanied *pari passu* by a great development of blood-vessels, and the uterus is thus supplied with blood sufficient not only for its own needs but also for the nourishment of the foetus. At the end of gestation the uterus presents the appearance of a large spheroidal body extending from the true pelvis nearly to the epigastrium.

Soon after the reception of the fecundated ovum the uterine **mucous membrane** undergoes proliferation, and forms the maternal envelopes known as the **deciduae**; these enter into close relation with the chorion or foetal envelope, and at delivery are in part extruded with the foetus.

The maternal envelope termed the **decidua vera** corresponds to the altered endometrium, except for the portion in which the ovum is actually embedded, and which when developed into the **placenta** constitutes a special bond of connexion between ovum and uterus. The decidua vera is produced by the proliferation of the endometrium, the glands becoming dilated and sacculated, and the vascular reticulum undergoing active development. The membrane attains its maximum proportions in the fifth month, when its thickness may amount to one centimetre. It consists of an external spongy layer, within which the uterine glands are transformed into cavities of irregular form, and of an internal compact layer interrupted only by a few glandular ducts. Its matrix is composed of blood-vessels and of round, elongated, and polyhedral cells, which are embedded in an amorphous ground-substance, and some of which are multinuclear and considerably larger than the cells of the endometrium in its ordinary condition. The epithelial cells lining the dilated uterine glands are generally cubical or flattened, some of them appearing swollen and in process of mucous or fatty degeneration. In the later stages of pregnancy the epithelium of the deeper-lying glandular cavities disappears altogether, in the more superficial cavities it persists throughout.

The **decidua reflexa** is formed by the proliferation of the decidua vera, which grows over the surface of the ovum from the margin of attachment of the latter to the uterine wall. This membrane accordingly resembles the decidua vera in its general structure. It is not until about the fifth month, when the increased bulk of the ovum brings the two membranes into contact, that they coalesce and the glands and blood-vessels of the reflexa disappear (LEOPOLD), the reflexa thenceforward receiving its blood-supply directly from the vera.

The **decidua serotina** is the part of the proliferous endometrium over which the ovum is attached to the uterus. Its structure resembles that of the vera, except for the fact that the vessels of the muscular and mucous layers are more abundant and larger.

The outer envelope of the ovum, or **chorion**, is within the first few weeks of gestation beset over its entire surface with vascular processes or villi, which insinuate themselves into the substance of the reflexa and serotina. With the growth of the ovum the reflexa becomes stretched, thinned out, and non-vascular, and the chorionic villi embedded in it also undergo retrograde changes, losing their blood-vessels and being converted into slender filaments. In the serotina, on the other hand, the proliferous endometrium grows out to meet and blend with the chorionic villi, which thus form what is called the **foetal placenta**. Where the tips of the chorionic villi come into contact with the mucous membrane the cells of the serotina grow round them, and a firm connexion is thus established between the two structures. At the same time the serotinal blood-vessels grow out toward the villi, the capillaries dilating and becoming closely applied to the capillaries of the villi, so that when the latter send out lateral buds or offshoots these readily break through the thin walls of the serotinal capillaries, and projecting freely within their channels are bathed in the serotinal blood (LEOPOLD).

The chorionic villi and the serotinal tissue continue their growth toward each other for some time longer, and the latter, especially over the spots corresponding to the limits of the cotyledons, sends long processes into the intervillous tissue. The chorion itself is however reached by these processes only at the edge of the placenta, where they overspread its external surface for some distance.

With the growth of the placenta the blood-spaces produced by the dilatation of the serotinal capillaries become ever wider, until at length when the placenta is fully developed (fourth month), they form one great sinus between serotina and chorion, imperfectly subdivided into minor sinuses by the villi and the serotinal processes alone. Into these sinuses blood enters from the thin-walled serotinal arteries, and after bathing the villi of the foetal placenta, returns partly through the large marginal vein of the placenta, and partly through veins, of the endometrium that open directly into the great sinus and its sub-divisions.

The surface of the chorionic villi is covered over with epithelium derived from the ovum, and it is only at the junction of the villi with the decidua that this epithelium disappears towards the end of gestation. The inner surface of the serotina bounding the blood-sinuses is covered with a layer of endothelial cells.

The portion of the serotina that takes part in the formation of the placenta is termed the **maternal placenta**. Its elaboration is always associated with an abundant development of blood-vessels throughout the subserotinal muscular stratum, but up to the end of gestation the vessels continue to be separated by endometrial tissue containing remnants of uterine glands.

As early as the fifth month multinuclear giant-cells make their appearance in the serotina and in the muscular tissue immediately underlying it. From the eighth month onwards some of the wide placental veins begin to be occluded by thrombosis, and it is probable that the ever-increasing impediment to the venous efflux thus created may be one of the factors which determine the onset of labour (LEOPOLD).

The separation of the placenta and the foetal membranes takes place within the spongy layer of the decidua, and accordingly the chorion and placenta when extruded as the **after-birth** are overlaid by a thin stratum of decidual tissue. The deepest layer of the endometrium, containing the bases of the uterine glands, remains in the uterus, and from it the normal structure is reproduced during involution.

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Bumm: Development of the maternal circulation in the placenta *A. f. Gynäk.* xliii **1892**
Colluci: *Porzio materna della placenta* Bologna **1886**; *Struttura della placenta umana* Bologna **1887**
Ercolani: *Della struttura anat. della caduca uterina* Bologna **1874**; Normal and morbid anatomy of the placenta *A. ital. de biol.* iv **1883**
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Leopold: *A. f. Gynäk.* xi **1877** xii **1878** xxxv **1889**
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332. The pathological changes that occur in the **decidual membranes** during gestation are not yet fully understood, though it is certain that in some cases the premature expulsion of the ovum is referable to lesions of the uterine mucous membrane. Many reports of cases have no doubt been published, in which morbid proliferation, inflammation, induration, fatty degeneration, etc., of the deciduae are referred to; but it is highly probable that in this connexion much that is described as morbid is really physiological and normal.

In the first place, the endometrium is liable to undergo excessive proliferation or **hyperplasia**, whereby the deciduae (and in particular the vera) become abnormally thick, the overgrowth being generally attributed to chronic decidual endometritis. The hyperplastic thickening is met with chiefly in ova that are expelled between the second and the fourth month of pregnancy, and either extends over the whole of the decidua vera or is local and partial, in the latter case assuming the form of nodose and polypous excrescences (*endometritis decidua, tuberosa*, or *polyposa*). As such overgrowths have been observed chiefly in women who before pregnancy were the subjects of chronic endometritis, it is probable that the conditions are related. The cause of the primary inflammation may be gonorrhoeal or syphilitic infection, or some other morbid irritation.

Not infrequently **haemorrhage** from the decidua takes place, either in connexion with hyperplastic thickening or independently. In many instances the bleeding is due to the death of the embryo and the separation of the ovum consequent thereon.

When the haemorrhage is recurrent and the effused blood coagulates, the solidified fibrin blends with the foetal membranes into a blood-soaked mass not unlike a lump of gory flesh: this is known as a thrombotic or **fleshy mole**. When the mass is expelled the foetus is sometimes found to be intact, though smaller than it should be for its age. In other cases however the foetus cannot be discovered, having been entirely absorbed (Art. 333), though this can happen only when at the time of its death it was still very small.

In the later stages of gestation the most important changes affecting the deciduae are those relating to the serotina or maternal **placenta**, as it is from this source that the foetus is nourished, and disorders of the placental tissues are thus the most likely to bring about premature expulsion of the ovum. The changes in question consist mainly in fatty or hyaline degeneration, calcification, thickening, and cellular infiltration of the decidual tissue, and in partial thrombosis of the maternal vascular sinuses. **Fatty degeneration** of the decidual tissue is often indicated to the naked eye by the presence of small white spots in the maternal placenta: in other instances the change can be detected only with the microscope. It chiefly or exclusively affects the decidual cells, which are sometimes completely broken down at the places marked by the white spots. The fatty change is not infrequently associated with **calcareous infiltration**, generally near the attachments of the chorionic villi.

Hyperplastic **thickening** of the placenta is usually of very limited extent, and is in most cases due to simple increase of the decidual portion of its tissue. Diffuse and nodose fibrous hyperplasia has been described by R. Maier.

Cellular infiltration is ordinarily distributed in scattered patches and tracts, the former at times reaching the size of a tubercle. Occasionally the centre of the cellular patch is occupied by curdy detritus.

Thromboses of the maternal sinuses (sometimes erroneously described as placental infarcts) appear either as rounded or irregular patches, from 2 to 30 or even 50 millimetres in diameter, and lying just beneath the decidua or within the foetal placenta, or as laminar plates applied to the outer surface of the chorion. They are usually yellowish-white or reddish-yellow in tint, like decolorised splenic infarcts, and this resemblance has led to the term 'white infarcts' being applied to them. The placental tissue about them is always firmer than elsewhere.

The subchorionic thrombi are dense and compact in texture, and consist of homogeneous fibrin, which is often distinctly stratified and traversed by clefts and channels (the canalised fibrin of LANGHANS). They usually enclose but few cellular elements. The subplacental thrombi frequently contain enclosures that are white and opaque, or rust-coloured and pulpy; and sometimes they are excavated into hollows filled with liquid, which in different cases is clear or turbid, colourless, or tinged yellow or brownish.

The mass of the thrombus is made up partly of compact hyaline or stratified fibrin, partly of fibrillar fibrin, enclosing varying proportions of white and red blood-corpuscles. At times the leucocytes are aggregated into masses, as if they had at some previous time been crowding into the placental sinuses. In the softer parts of the mass the fibrin is granular, and interspersed with the amorphous or crystalline detritus of red corpuscles, or occasionally mingled with recently-extravasated blood.

The placental villi enclosed within the fibrinous masses have in most cases lost their epithelium. At the margin the nuclei of the connective-tissue cells are still capable of being stained with reagents and so rendered visible. In the interior of the larger thrombi the tissue of the villi looks cloudy or homogeneous, the nuclei no longer take up stains, while the vessels are impervious and often cease to be traceable. The villi are therefore manifestly necrotic, and at times they are even calcified. The overlying decidua may be unaltered or beset with patches of cellular infiltration. Occasionally the intercellular ground-substance appears to be increased, and then looks not unlike the hyaline stratified or fibrillar fibrin of the thrombi. The cells enclosed in it are sometimes intact, sometimes necrotic and denucleated.

The above-described changes are certainly not all of a pathological nature. Thus, for example, fatty degeneration of the decidual tissue at the end of gestation is so common, that it is only when it is excessive or premature that we can regard it as of any importance. And even thickening of the decidua is in most cases evidence merely of some trivial variation or idiosyncrasy in relation to the development of the serotina or the mode of separation of the placenta. In estimating the thickness of the decidua it must also be borne in mind that the serotina at the margin of the placenta is considerably deeper than at the centre.

Cellular infiltrations and fibroid changes are of greater significance. They appear for the most part in the foetal membranes of syphilitic mothers, though at present there are no sufficient grounds for deciding definitely that they are necessarily of syphilitic origin.

Subchorionic coagula are, according to LANGHANS, of constant occurrence in the last months of pregnancy, and only when they are abnormally large can they be regarded as morbid.

Large subdecidual thrombi appear to be specially common in women affected with syphilis; but it is beyond doubt that they are also met with in mothers who are not syphilitic, for example, in those who suffer from Bright's disease. When such thrombi are associated with cellular infiltrations, their formation is perhaps referable to some inflammatory process; but in the absence of infiltration it is not easy to connect thrombosis with any of the other textural changes that have been observed. Probably the most natural supposition is that the coagulation is due to wide-spread occlusion of the efferent veins. In other cases it may be induced by some primary alteration in the blood; or where morbid changes in the villi have taken place, which from their nature must have been antecedent, these changes themselves may be regarded as sufficient to produce thrombosis.

References on Morbid Changes in the Deciduae (see also Arts. 333 and 334).

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FEHLING: Habitual abortion in women with renal disease *A. f. Gynäk.* XXVII **1886**
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MAIER: Fibroid changes in the placenta *Monatsschr. f. Geburtsh.* XXXII and *V. A.* 45 **1869**
ROHR: White infarcts *V. A.* 115 **1889**
ROSSIER: White infarcts *Inaug. Diss.* Basle **1888**
SLAVJANSKI: Decidual endometritis *A. f. Gynäk.* IV **1872**; Syphilis *Prager Vierteljahrsschr.* 109 **1871**
VIRCHOW: Syphilis *V. A.* 21 **1861**; *Die krankhaften Geschwülste* II Berlin **1865**
WINKLER: *Die Adnexen d. menschlichen Eies* Jena **1870**
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333. The **foetal membranes** consist of the amnion and chorion, the latter going to form the foetal placenta, which receives its blood-vessels from the umbilical cord.

The **amnion** consists of a thin fibrous membrane and an internal layer of epithelium, which lines the inside of the embryo-sac and is continued over the cord.

The **chorion** forms the external envelope of the embryo, and is lined on its outer surface with epithelium; it very soon acquires a shaggy coat of villous processes, which are furnished with capillaries derived from the umbilical vessels. As development proceeds the villi grow luxuriantly over the part where the placenta is formed (*chorion frondosum*), while everywhere else they dwindle and disappear (*chorion laeve*).

By the end of gestation the **placenta** appears as a cake-shaped organ of about 3 centimetres in thickness and 14 to 16 centimetres in width, and weighing 500 grammes or more. The **foetal placenta** is made up of the amnion and the superimposed chorion with its dendritic, vascular, and epithelium-covered villi, which are in contact externally with the **maternal placenta.** The villi are separated by deep furrows, into which pass processes of the maternal placenta, these processes being grouped into lobes known as **cotyledons.**

It is not yet certainly determined whether as a matter of fact the foetal envelopes are liable to defects of development, and whether, if such imperfection actually occurs, it or primary **degeneration of the chorion** and its villi has anything to do with the abortions that are so common in the first months of gestation.

In particular cases of abortion during the early months the villi have been found to be remarkably small. Moreover, when the ovum has been prematurely expelled, owing to the death of the embryo, to decidual disease, or to haemorrhage, the villi and their epithelium are sometimes found to have undergone fatty or mucous degeneration, or to be overlaid with coagulated blood. Not infrequently the blood effused in successive haemorrhages clots into a solid mass, which in some parts is red (from the presence of red corpuscles), in other parts yellowish or greyish-white, and which cannot be separated from the foetal envelopes without tearing off the villi: this mass constitutes the thrombotic or **fleshy mole** described in Art. 332.

Hypertrophy and **gelatinous degeneration** of the chorionic villi may supervene in the earlier or the later months of gestation, and may extend over the whole surface of the ovum or be limited to particular portions of it, affecting for example only the villi of the placenta or of one of its cotyledons.

The hypertrophied villi are abnormally large, and many of their twigs are swollen and flask-shaped. When they consist of fibrous tissue the resulting structure might be described as a **fibroma** of the placenta. When mucous degeneration supervenes the twigs swell to a still larger size, and become club-shaped, spindle-shaped, and globular, so that they come to resemble a bunch of thin-skinned grapes or thin-walled bladders (Fig. 536). The structure so produced is accordingly described as a vesicular, hydatidiform, or briefly **hydatid mole.** VIRCHOW classes such moles with the **myxomata**.

The separate vesicles measure 2 to 12 or more millimetres in diameter, and hang from slender pedicles attached to other vesicles or to the chorion itself. Their substance consists of mucous tissue with a few cells and fibres, whose wide interstices are occupied with liquid containing mucin. When the degeneration sets in early the fibrous elements are scanty; but if the chorion is already somewhat advanced in development before it degenerates the texture is more like that of oedematous fibrous tissue.

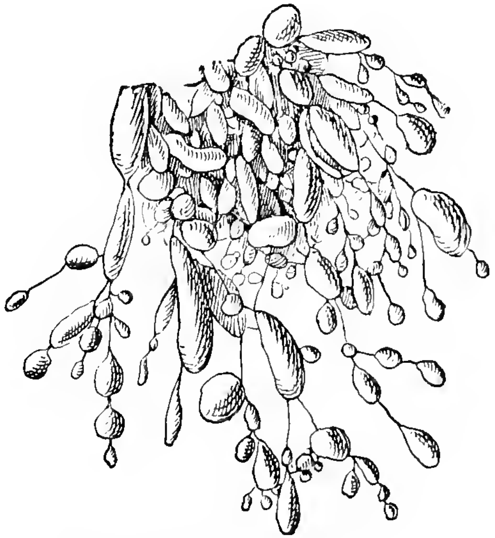

FIG. 536. VILLI OF A HYDATID MOLE.
(*Natural size*)

The cause of this peculiar overgrowth and metaplasia of the chorionic villi is still uncertain. Sometimes the mucous degeneration affects portions only of the chorion, or is limited to the umbilical cord, the villi themselves being exempt.

If the degeneration of the villi is at all wide-spread the embryo generally perishes, and in early abortions is apt to be completely absorbed. Even when the embryo dies, however, the foetal membranes remain for some time in the uterus, and continue to grow, provided they are adequately supplied with blood from the deciduae. When the degeneration of the membranes is local, the foetus may either become malformed and stunted, or develope normally. When two ova are present in the uterus at the same time, one may develope normally and the other be converted into a hydatid mole.

The tips of the degenerated villi are in some cases free, in others attached to the uterine mucous membrane, and are occasionally so firmly adherent to the latter that they remain behind when the membranes are extruded. Cases have moreover been recorded (VOLKMANN, JAROTZKY, WALDEYER, KRIEGER, and MEYER) in which these structures had undergone proliferation and penetrated deeply into the uterine wall (Art. 334, Fig. 539).

Haemorrhage from the decidua is very common in cases of hydatidiform degeneration, and gives rise to various combinations of the hydatid with the fleshy mole.

Among the morbid changes of the **foetal placenta**, other than those related to the formation of moles, fatty, calcareous, and necrotic degenerations of particular villi are the commonest. Necrosis is usually a result of the thrombosis of the placental sinuses above described. Fatty and calcareous changes other than necrotic are very frequently observed, and indeed the appearance of fat in some of the cells of the chorion or its villi towards the end of gestation can hardly be regarded as pathological. Fibrous thickening, taking the form of somewhat elevated patches in the placental chorion, is also of normal occurrence.

Inflammatory changes indicated by cellular infiltration, disseminated or diffuse, are met with in the chorion, in the placental villi, and in the umbilical cord; they generally follow the course of the vessels. In the placental chorion the infiltration sometimes extends beyond the fibrous layer to the external epithelial stratum, which according to LANGHANS is normally liable, towards the end of pregnancy, to be transformed into hyaline canalised fibrin.

When such inflammatory changes are present the villi appear thickened and hyperplastic; but where the evidence of inflammation is slight it is not easy to say whether a villus is really hypertrophied or not.

In some cases of inflammation of the chorion and umbilical cord the vessel-walls of these structures also show signs of cellular infiltration (Fig. 537 *c*) or fibroid induration; in exceptional instances some of them are calcified, and white or mixed thrombi are apt to be deposited on the altered intima.

The last-named changes are in general associated with syphilitic lesions of the foetus and mother; the infiltrated and indurated vessel-walls sometimes indeed enclose necrotic foci (Fig. 537 *c*), and the structure thus produced might fairly be described as gummatous. Under like conditions wide-spread hyperplasia of the placental villi is said to take place in certain cases (FRÄNKEL).

Tuberculosis of the placenta is rare, though cases have been observed in which the chorionic villi contained typical tubercles (LEHMANN, SCHMORL, KOCKEL).

As examples of variation affecting the **size and form** of the placenta we may mention excess or defect in regard to its volume, its subdivision into from two to seven *placentulae*, and its deformation into the shape of a horse-shoe when it is attached near the internal os uteri. Not infrequently it assumes the form known as **placenta marginata**, in which at a little distance from its border it appears encircled more or less completely by a white band, consisting of decidual tissue, chorionic villi, fibrin, and calcareous deposits (O. KÜSTNER). In very rare cases no definite placenta is developed at all, the entire surface of the ovum being attached by its vascular villi to the decidua.

The length of the **umbilical cord** varies within wide limits, the maximum being about 190 centimetres; on the other hand it may be so extremely short that the placenta seems directly attached to the navel. Usually the cord is inserted about the middle of the placenta, but not infrequently it arises from the edge (battledore placenta or *insertio marginalis*). In rare cases the insertion is extra-placental, the blood-vessels entering the chorion and running along its surface to the placenta (*insertio velamentosa*). One of the two umbilical arteries may be absent, though this is uncommon (Fig. 537).

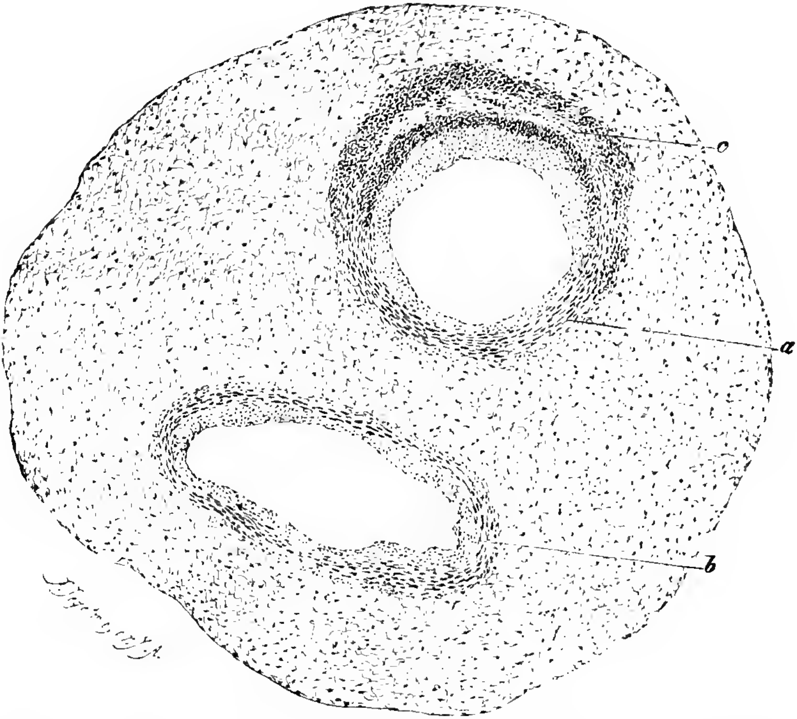

FIG. 537. SECTION OF THE UMBILICAL CORD OF A SYPHILITIC INFANT WITH A SINGLE ARTERY.

(*Preparation hardened in Müller's fluid, and stained with haematoxylin*: × 8)

a artery *b* vein *c* syphilitic (gummatous) deposit

Knots and twists are frequently formed in the cord, but they seldom interfere with the circulation to such an extent as to cause the death of the foetus. The twists that are met with in the case of still-born foetuses have usually been produced after death (Ruge, Martin). The cord is very often looped round some part of the body, but this seldom results in the death or the mutilation of the foetus.

Hydramnios is an excessive accumulation of liquid (*liquor amnii*) within the membranes, the weight of which by the end of pregnancy may considerably exceed 1·5 kilogrammes. Two varieties, an acute (Charpentier) and a chronic, have been distinguished. The chronic form is associated with oedematous or dropsical conditions of the mother, or results from hypertrophy of the placenta, thickening of the deciduae, and persistence of the vessels of the non-villous portion of the chorion (Jungbluth) which normally become obliterated. The foetus may be well developed or stunted; if the hydramnios is large the foetus may die.

The acute form generally makes its appearance between the fourth and the sixth month (Schroeder), and sometimes within a week or two distends the uterus beyond the size usual at the end of pregnancy. The cause of this condition is unknown.

Toward the end of pregnancy the amnion is liable to rupture, and separate itself from the chorion. The movements of the foetus in some cases give rise to the formation of bands or infoldings which indent or constrict the umbilical cord (Braun, Lebedeff). According to Ahlfeld, strips of the amniotic epithelium are very often scratched off the membrane by the nails of the full-time foetus as it moves about. When the liquor amnii is scanty the amnion is apt to be thrown into folds and to form abnormal attachments to the skin of the foetus; these give rise to local malformations of the adherent parts.

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334. The **separation of the ovum** in parturition takes place at the spongy layer of the decidua vera and serotina, the proportionate thickness of the portions of the membrane respectively retained and expelled being subject to considerable variation. The portion retained consists of the cellular tissue of the deepest layers of the endometrium with the bases of the glands, whose epithelium persists up to the end of pregnancy.

Immediately after delivery **regenerative proliferation** of the mucous membrane sets in, though in the first few days of the puerperium fragments of the superficial layers continue to be shed and a certain amount of blood continues to ooze from the vessels, with the result that the hyperaemic mucous membrane appears for some time to be smeared with liquid or clotted blood, and with partially-exfoliated greyish-white or yellowish-white shreds of fatty mucous membrane. At the same time cells migrate from the blood-vessels, and mingling with the cast-off shreds and the extravasated blood are discharged from the uterus as the **lochia**.

Both at the site of the placenta and beyond it a new endometrium with its glands is produced. Over the placental site the organisation of the thrombi in the uterine sinuses, which had begun before labour set in, proceeds rapidly, and the large thrombosed veins that after delivery project from the raw surface grow smaller by degrees and sink back into the thickness of the uterine wall.

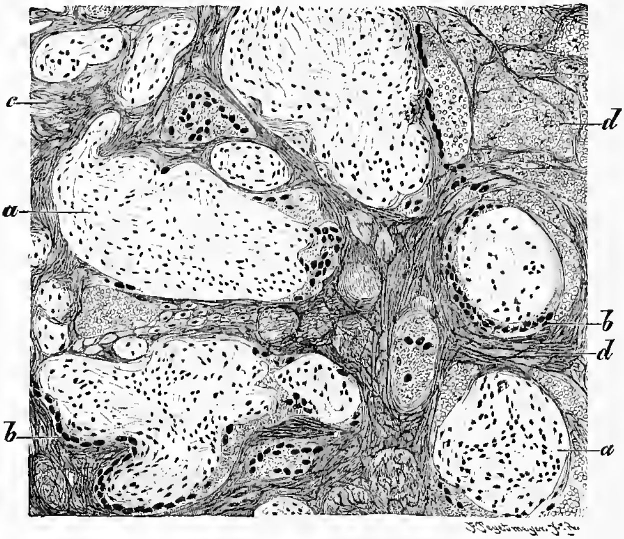

Fig. 538. Placental polypus of six weeks' standing.

(Preparation hardened in Müller's fluid, stained with haematoxylin, and mounted in Canada balsam: × 45)

a placental villi *b* epithelial cells *c* fibrin *d* red blood-corpuscles

In the course of four to six weeks the regeneration of the mucous membrane is usually completed, though the rate at which it proceeds varies greatly in different cases, and is liable to be considerably retarded by various causes, such as general weakness of the constitution or tuberculosis (Kundrat, Leopold).

Until the process of regeneration has reached a certain stage of completeness, the uterine surface between the mouths of the glands must be regarded as if it were an open wound, inasmuch as it is not covered with epithelium, and the lymphatics and blood-vessels are thus exposed and unprotected.

When the ovum is expelled, whether prematurely or at full time, it not infrequently happens that fragments of the foetal envelopes or of the placenta remain in the uterus, and that the decidua is imperfectly detached and extruded. In most cases these membranes and decidual remnants are afterwards expelled or evacuated artificially, but they occasionally remain within the uterus for weeks or even for months, and give rise to ulterior disturbances.

The most common result of any importance is the supervention of **haemorrhage** and the formation of **fibrinous polypi** (decidual, chorionic, or placental) or uterine haematomata, the effused blood undergoing partial coagulation, and so permeating and enclosing in masses of fibrin the remnants of the decidua, of the membranes, or of the placenta (Fig. 538 *a b c d*). When the haemorrhage is recurrent the haematoma sometimes attains the size of the fist, and at length projects into the cervix. In cases of some standing the outer layers of the polypus are remarkably firm, while the interior parts may be partially softened. The base is adherent to the mucous membrane.

According to KÜSTNER, CHIARI, SCHMORL, GOTTSCHALK, SÄNGER, and others, after the premature expulsion of an apparently-normal ovum, or after the removal of a hydatid mole, retained fragments of the decidua serotina occasionally undergo proliferous changes, giving rise to diffuse or polypous hyperplasia of the membrane, or even to an actual neoplasm, which has been variously described as **deciduoma malignum** or **sarcoma deciduo-cellulare** (SÄNGER). This growth sometimes spreads superficially and sometimes assumes the form of rounded nodes: in either case it rapidly produces metastases in the uterus and elsewhere. The original tumour and its metastases are essentially of an epithelial nature (MARCHAND), the cells lining the chorion (syncytium and ectodermal layer) undergoing neoplastic proliferation, and producing large multinuclear and polyhedral cells. The intrauterine growth gives rise to profuse haemorrhage and foetid watery discharges. The malignancy of the neoplasm is extreme, and the patient speedily becomes cachetic and dies, unless the uterus is removed in time.

In rare cases remnants of the placenta also become proliferous, and grow into an autonomous malignant neoplasm, that infiltrates the uterine wall by way of the blood-vessels (Fig. 539 *e f g*), and thus by interfering with the circulation induces necrosis and disintegration of the invaded tissues. Such growths are accordingly described as destructive placental polypi or **malignant placentomata.** The neoplastic proliferation appears in general to start in degenerate chorionic villi (Art. 333), but it is also liable to originate in placental remnants that have not undergone myxomatous change. Papillary outgrowths are produced that resemble chorionic villi (Fig. 539 *d* d_1 *e f*), some being composed merely of aggregated epithelial cells, while others also enclose a certain amount of delicate fibrous tissue.

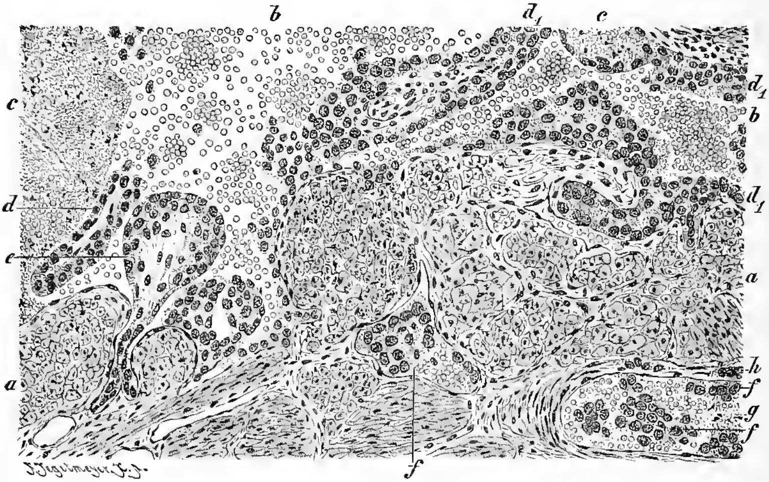

FIG. 539. DESTRUCTIVE PLACENTAL POLYPUS AT ITS ATTACHMENT TO THE UTERINE WALL (VON KAHLDEN).

(Preparation hardened in Müller's fluid, embedded for section in celloidin, and stained with haematoxylin : × 70)

a	muscular tissue of the uterus
b	large venous sinus
c	thrombus
d d_1	intravascular ingrowths from the chorionic villi into a large blood-sinus, some lying free (*d*), others attached to the sinus-wall (d_1)
e	proliferous villus penetrating into a small vessel
f	proliferous chorionic epithelial cells within the veins of the uterine wall
g	thrombus
h	proliferous cells in the wall of a vein

According as the epithelial or the fibrous elements predominate in these neoplastic villi, the growth may be classed as a carcinoma or as a sarcoma.

Combinations of haematoma with overgrowth of the decidua or of the placental villi are described.

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335. **The puerperal state.** By the act of parturition conditions are usually induced, both in the uterus and in the cervix and vagina, which render these parts highly susceptible to **septic infection,** the most important exciting agents being the pathogenic micrococci. The commonest injuries are bruises and lacerations of the cervix and the vaginal orifice, and these must in fact be regarded as unprotected wounds. When infective micro-organisms gain access to the genital canal immediately before or after delivery, the chances of traumatic septic infection are very great indeed, and the result is often enough the induction of **inflammation,** purulent, phlegmonous, diphtheritic, pyaemic, or septicaemic as the case may be. The infection commonly starts in wounds of the vulva, vagina, and cervix, these parts being more exposed than the uterus, though the first local manifestation may appear in the interior of the uterus, whose secretions afford a suitable nidus for the settlement and multiplication of microbes.

The infected lacerations of the vagina and cervix are frequently converted into ulcers, whose margins and floors are somewhat densely infiltrated with leucocytes, and ultimately suppurate, slough, or become gangrenous; but from such an infected wound of the vagina it is possible for grave general infection to proceed, though no suppuration or gangrene is apparent about the local lesion itself, and the like is true of lacerations and contusions of the cervix.

Infection of the interior of the uterus is usually followed by putrid decomposition of the extravasated blood and exfoliated decidual tissue, the surface assuming a dirty-grey, greyish-yellow, brown, or black colour, and giving off a foetid odour. More or less extensive inflammation of the mucous membrane is set up, and issues in suppuration, sloughing, or gangrene. Sometimes the surface is covered over with a film of croupous exudation.

No rule can be laid down as to the ultimate extension of these destructive processes. The site of the placenta may be involved at an early stage, or it may remain for a long time unaffected.

In the milder cases the inflammation is limited to the part first infected, though it frequently spreads widely, both in depth and extent. Infection proceeding from a vulvar laceration spreads to the vagina, the pelvic cellular tissue, and the labia, and leads to more or less considerable swelling of these parts, which is due to inflammatory oedema, often accompanied by haemorrhagic or purulent infiltration: here and there this oedema is apt to result in suppuration or even in gangrene. Infection proceeding from wounds of the cervix spreads to the underlying cellular tissue: that proceeding from the uterine surface invades the underlying muscular wall, and the inflammation frequently extends to the broad ligaments and the serous covering of the uterus.

The infection travels by way of the wide interstitial lymph-spaces of the uterine wall, whose tissues after parturition are relaxed and flushed by an abundant stream of lymph. The inflammatory swelling of the parametric cellular tissue and of the uterine wall itself accordingly tends to become diffuse: at times however only certain tracts of the tissue appear to be involved, the infection extending along particular lymphatics or blood-vessels. In these cases the uterine wall or the pelvic cellular tissue contains lymphatics filled with pus, or veins with softened and puriform thrombi. The parametric inflammation occasionally spreads very widely, passing up beneath the peritoneum as far as the perinephric tissue or over the brim of the false pelvis down to the thighs. The path taken by the inflammation is indicated by serous, haemorrhagic, and purulent oedema, abscesses, gangrene, and pyaemic thrombosis of the blood-vessels and lymphatics.

When the inflammation is extensive the pelvic peritoneum, and often the abdominal peritoneum also, are sooner or later involved; even the pleura, pericardium, and interlobular pulmonary tissues are sometimes affected, and fibrinous, fibrino-purulent, sero-purulent, or putrid exudations are thus effused into these tissues.

In inflammation of the broad ligaments and the pelvic peritoneum, the ovary always becomes more or less inflamed and swollen, and in some cases is partially or completely destroyed by suppuration or gangrenous softening and putrefaction. The tubes are generally swollen and infiltrated, the inflammation passing to them from the peritoneum. It is only in very rare cases that puerperal endometritis spreads from the uterus directly to the tubes, and thence to the peritoneum.

The system generally is always more or less affected in these varieties of infection, toxic substances elaborated at the local seat of inflammation being taken up and diffused by the lymphatics and blood-vessels. The septic microbes themselves are also liable to gain entrance into the circulation, and set up metastatic inflammations in distant parts.

When the result is not fatal, the exudations are re-absorbed or break through into the adjacent hollow viscera and so escape. Induration of the pelvic cellular tissue, fibrous hyperplasia of the uterus, pelvic adhesions, and at times atrophy of the endometrium, are the ordinary after-effects. Puerperal endometritis not issuing in destruction of the mucous membrane is occasionally followed by chronic inflammation and polypous hyperplasia. In the parametritic indurations residual colonies of the pathogenic microbes sometimes persist, in a living though quiescent condition, for a long period of time.

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336. In **extra-uterine gestation** or ectopic pregnancy the ovum developes outside the uterus. It is distinguished according to the position of the ovum as abdominal or tubal: but it is highly probable that all abdominal pregnancies are at the outset tubal.

The conditions for the existence of an ectopic pregnancy are that the ovum shall be fecundated outside the uterus, and that it shall then be prevented from migrating into the uterine cavity. As the ovum developes in its false position, a decidua similar in structure to the normal decidua vera is formed within the uterus from the endometrium.

In **abdominal pregnancy** the ovum is found within the abdominal cavity and becomes adherent to the peritoneum; in some cases however it remains perfectly free, excepting over the site of the placenta. According to LANGHANS and WALKER, the pelvic peritoneum forms a maternal envelope of connective tissue for the ovum, about which cellular proliferation takes place, producing a structure somewhat resembling decidual tissue. The development of the foetus may advance to full time, whether the membranes continue intact or by rupturing allow it to escape into the abdominal cavity. At the end of gestation the foetus dies, owing to the inevitable interference with the placental circulation, and the decidua of the uterus is thereupon extruded. In other cases the foetus dies before the end of the normal term.

When the dead foetus undergoes decomposition, it acts as an irritant on the surrounding tissues, often setting up suppurative inflammation, which is followed either by fatal peritonitis, or by irruption of the pus into an adjacent hollow viscus or through the abdominal wall to the exterior. After the decomposed foetus is extruded, either entire or in successive portions consisting of bones, putrid flesh, and pus, recovery and repair may take place in the course of months or years; but a fatal issue is more usual. When the foetus produces little or no irritation, it may remain within the abdominal cavity for an indefinite time.

Tubal pregnancy is the commonest and by far the most important form of extra-uterine gestation, three varieties being distinguished according to the place occupied by the ovum, namely tubal (in the narrower sense), tubo-abdominal, and tubo-uterine or interstitial.

According to LANGHANS and LEOPOLD, no decidua reflexa is formed in tubal pregnancy, but the mucosa surrounding the ovum is converted into decidual tissue. As the foetal sac enlarges the muscular fibres of the tube-wall are pushed asunder, and the sac, the greater part of which is covered only by peritoneum, projects into the abdominal cavity. In certain cases it intrudes to some extent between the layers of the broad ligament.

In rare cases the ovum may develope within the tube up to the normal term; but in general the sac ruptures at some time between the first and the fifth month, giving rise to haemorrhage that is often fatal. If the haemorrhage is not fatal the effused blood is partially re-absorbed, but it usually induces inflammation of varying intensity and extent, which in its turn gives rise to adhesions between the pelvic and abdominal viscera. The foetus may escape from the tube with or without its membranes.

In tubo-uterine gestation, in which the ovum developes within the uterine portion of the tube, the sac usually gives way in like manner during the early months of pregnancy, and the foetus either enters the abdominal cavity or remains embedded among the fibres of the uterine wall. In very rare cases the ovum subsequently escapes into the uterus.

In tubo-abdominal cases the ovum is only in part embraced by the abdominal end of the tube, but in other respects behaves as in genuine tubal pregnancy.

Ovarian pregnancy, in which the foetus is said to develope, and even to advance to the normal term, within one of the graafian follicles, has been described by some authorities of weight. But the evidence for its actual occurrence is so inconclusive, and the theoretical difficulties in regard to its possibility so great, that its existence cannot be regarded as even probable. In certain cases at least an ovarian dermoid has been mistaken for the remains of an ovarian pregnancy.

In all cases of extra-uterine pregnancy the embryo, if it dies while still small, is liable to be re-absorbed. If it does not die until a comparatively late stage in its growth, and is not extruded in the manner above described, it remains permanently within the body, and is sometimes carried for many years. It either retains some semblance of its shape, and is swathed in a kind of fibrous mantle, or it is macerated into a pulpy mass containing fragments of bone, fat, cholesterin, and pigment, enclosed in a capsule of connective tissue. Sooner or later it is usually calcified, and converted into a **lithopaedion** or petrified mummy. According to KÜCHENMEISTER, three varieties of lithopaedia are distinguishable. In the first the mummified foetus can easily be enucleated from the calcareous membranes (*lithokelyphos*). In the second the foetus while still alive has become adherent to the membranes at several points. Later on the agglutinated parts are calcified, while others become dry and mummified (*lithokelyphopaedion*). In the third variety the foetus, by the bursting of its sac, escapes into the abdominal cavity, and becomes subsequently infiltrated with calcareous salts (true *lithopaedion*).

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CHAPTER CX

THE MAMMARY GLANDS

337. The **mamma** of the new-born infant, which is at most 2 centimetres wide and 1 centimetre thick, is an organ whose glandular elements consist of ten to twenty and more lactiferous ducts, most of them opening into one or other of the pitted depressions of the nipple. The ducts are lined with columnar or stratified squamous epithelium, and in both girls and boys some of them are distended by masses of desquamated epithelium, granular detritus, and liquid. For a few days after birth some of this liquid can generally be pressed out, and is often described as milk. According to KÖLLIKER, the lactiferous ducts continue to dilate during the first week, and the dilatation is in some cases so extreme that the whole gland acquires a cavernous structure: towards the middle of the first year the sacculations disappear. The glandular ducts are then invested throughout with columnar epithelium, they possess but few lateral branches, and end in rounded bulbs.

At the beginning of the twentieth year, by which time the lactiferous ducts send out lateral branches, subdivide at their ends, and acquire terminal acini, the gland in the male reaches its highest development. The widest diameter of the gland is 4 to 5 centimetres in length. Numerous cases are however on record in which the breast attained to still greater development, generally in connexion with pseudo-hermaphroditism. From the fiftieth year onwards the glandular acini and certain of the lactiferous ducts disappear, while others become dilated.

The fully-developed breast in nulliparous women is made up chiefly of coarse fibrous tissue, containing few cells, and of almost tendinous texture. Few of the ducts possess terminal vesicles or acini, and the lobules are small, ill-developed, and limited to the deepest parts of the gland. The growth of the glandular parenchyma during puberty is therefore slight. The terminal acini and lateral ducts are provided with a structureless basement-membrane and low columnar epithelium. With the onset of pregnancy the lactiferous ducts send off numbers of new buds and sprouts, which develope into lateral ducts and acini. At the time of lactation the gland attains its complete evolution, the fibrous tissue becoming soft and lax, and enclosing great numbers of large well-formed lobules. These lobules are situated mainly in the deeper parts of the gland and in the offshoots which it thrusts into the surrounding fatty tissue. The epithelium of the ducts and acini is columnar, but becomes more or less flattened out as the secretion accumulates. With the cessation of lactation the acini and therefore the lobules dwindle once more, while the interlobular fibrous tissue again increases. In old age the acini and lobules disappear completely, until at last nothing remains but the ducts.

Congenital absence of one breast is very rare. On the other hand, supernumerary nipples (polythelia) and supernumerary mammae (hypermastia or polymastia) are by no means uncommon, either in men or women.

The **supernumerary glands** are generally situated below and internally to the main gland, or in the axillae; cases are recorded in which they occupied the middle line of the abdomen, or were seated over the acromion and even on the thigh. In polythelia the accessory nipples are either situated on an ordinary gland or receive the excretory ducts of supernumerary glands.

Morbid changes in the mammary gland, other than those due to new-growths, usually make their appearance at the time of its greatest development and greatest functional activity, in other words, during pregnancy and lactation.

In suckling mothers inflammatory swelling and fissuring of the **nipple** are common, and lead to the formation of small cleft-like **ulcers**: they are produced by the efforts of the child in sucking. When the child suffers from **thrush** an aphthous film is apt to appear on the nipple; and **erysipelas** occasionally starts from an infected fissure.

Eczema and **syphilitic ulcers** of the nipple are met with not only in pregnant and suckling women, but in others also. Some of the syphilitic lesions are primary (initial sclerosis, hard chancre), others are secondary (mucous patches and moist papules).

The secretion of the breast is normally produced only after pregnancy has begun; but several cases have been noted (BEIGEL) in which repeated suction of the nipple by an infant has led to the secretion of milk by non-lactating women of various ages. In rare cases the mammae at the time of puberty produce small quantities of a milk-like liquid. The secretion of milk by the male breast has more than once been observed.

When one of the ducts of a milk-secreting breast is occluded or partially obliterated by some previous disease, the part of the duct behind the obstruction occasionally but not very frequently dilates into a milk-containing cyst, known as a milk-cyst or **galactocele**. It does not usually lead to inflammation of the surrounding tissue; but in some cases changes take place in the milk, and these excite inflammation and proliferation in the fibrous stroma. According to KÜSTNER, the mammary tissue may even soften and break down into pulpy detritus from such inflammation.

The most important puerperal affection of the breast is mammary inflammation or **mastitis.** It may follow on disorders of secretion resulting from disease of the nipple, and associated with lacteal retention and engorgement. Such engorgement does not however by itself induce inflammation, which is generally dependent on infection (by *Staphylococcus* or *Streptococcus pyogenes*) starting from mammillary ulcers and fissures and extending to the breast by way of the lymphatics. In particular cases the exciting agent probably passes along the lactiferous ducts, and setting up decomposition in the contained milk thereby gives rise to inflammation. The inflammation as a rule produces painful circumscribed swellings, and but seldom induces diffuse enlargement of the breast from inflammatory oedema.

Having reached a certain degree of intensity, the mastitis generally subsides; but in many cases it ends in more or less extensive suppuration and **mammary abscess.** When such an abscess ruptures and its pus is evacuated, healing usually takes place by granulation and cicatrisation: sinuses and fistulae however often remain behind, which can be closed only by appropriate surgical means. Local but somewhat ill-defined patches of induration are also apt to persist at the seat of the abscess.

Suppurative inflammation of a similar kind, involving the tissue in front of or behind the gland, occasionally supervenes during the puerperal period: it is described as **paramastitis.** Inflammation of the breast, apart from pregnancy and the puerperal state, is on the whole of rare occurrence. Acute mastitis, which may issue in suppuration, is not unknown in new-born infants: it gives rise to swelling and redness of the gland. At the time of puberty, moreover, somewhat painful but generally transient inflammatory swelling is met with, but it seldom leads to suppuration.

Tuberculosis of the breast is not very common. Caseous nodes, abscesses, and fistulous sinuses, surrounded by indurated cicatricial tissue, are the ordinary lesions it produces. Sometimes however the gland is studded with numberless grey and white tubercles, and with larger caseous nodules and softened patches. Certain **cold abscesses** of the breast are probably referable to tuberculosis. The tuberculous nodules are in some cases seated in the lobules, in other cases they beset the ducts. Syphilitic **gummatous mastitis** is rarely met with.

In the breasts of elderly women it is not uncommon to find small or moderate-sized **simple cysts.** They arise from the dilatation of the ducts, and their contents consist of limpid or viscid liquid, greenish, brownish, or yellowish in colour, and mingled with granular detritus, fatty crystals, and cholesterin: now and again they enclose a cream-like or buttery mass. The wall of the cyst at times undergoes calcification.

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338. Hyperplastic overgrowth of the mammary tissue is of more frequent occurrence than any of the changes above described. It gives rise either to general enlargement of the gland, or to the formation of circumscribed nodes in its substance. At puberty, in the first place, both breasts may undergo excessive development, from overgrowth of their normal constituent elements: the enlargement so produced is accordingly a true **hypertrophy** of the glands. It is only in rare instances that the enlargement is extreme, but examples are on record in which each of the breasts weighed from 4 to 7 kilogrammes. The structure of the hypertrophied breast corresponds to that which is normal

in young virgins; but when pregnancy ensues the ordinary process of evolution is set up, the glandular parenchyma becomes more abundant, and the structure comes to resemble that which is characteristic of the gravid state. So far as our present knowledge extends, it would appear that the overgrowth, provided it is really hypertrophic and not neoplastic, tends to cease after a time, the gland thenceforward remaining unchanged.

Of the **tumours** of the mamma, **adenoma** is nearest akin to simple hypertrophy. This growth, like the normal gland, is made up of a fibrous stroma (Fig. 540 *c* and Fig. 541 *c*) and glandular substance (*a b*). One difference between hypertrophy and adenoma consists in the fact that the tumour is unilateral and forms nodes, and that at the outset it involves only a segment of one gland. The glandular portion of its structure is composed mainly either of acini (Fig. 540 *a*) or of tubular ducts (Fig. 541 *b*) lined with columnar epithelium; and the two forms are accordingly distinguished as acinous and tubular adenoma respectively. The acini in the former variety are often very numerous (Fig. 540 *a*), but they do not form typical lobules like those of the normal gland; it is the production of new epithelial cells in the interior of the acini and ducts that is excessive rather than the elaboration of glandular elements. In tubular adenoma (Fig. 541) some of the glandular tubes are distributed uniformly through the stroma; others again are aggregated into groups lying side by side, owing probably to

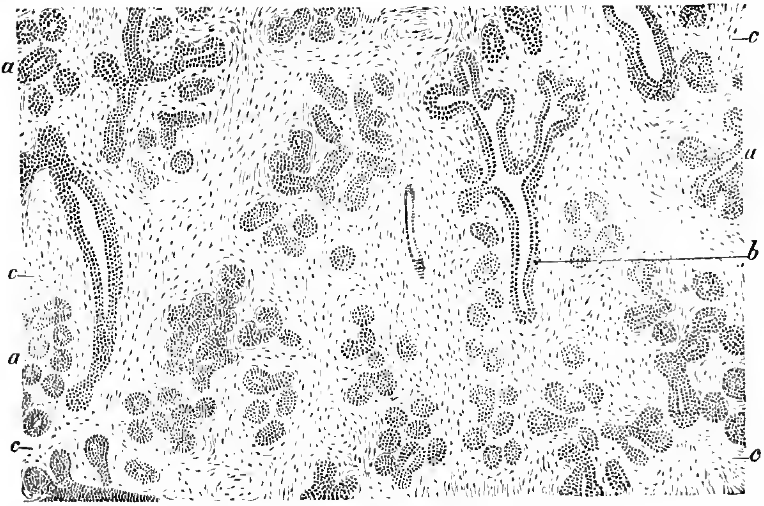

FIG. 540. ACINOUS ADENOMA OF THE BREAST.

(Preparation hardened in Müller's fluid and alcohol, stained with alum-carmine, and mounted in Canada balsam: × 30)

a acini *b* ducts *c* fibrous stroma

the fact that the neoplastic tubules originate from some particular glandular duct.

In both forms of adenoma the stroma is usually looser and more cellular than that of the normal gland. When it is relatively abundant the tumour might be described as an **adenofibroma** or fibro-adenoma. Should the fibrous elements in an adenofibroma become excessively developed, while the neoplastic overgrowth of the glandular elements is relatively inconsiderable, the tumour approximates by degrees to the histioid type. There is thus a graduated series of **connective-tissue tumours** which are related to the adenofibromata, those most akin to the latter in structure still containing neoplastic glandular elements, while those that are most remote consist essentially of non-glandular fibrous or fibro-cellular tissue.

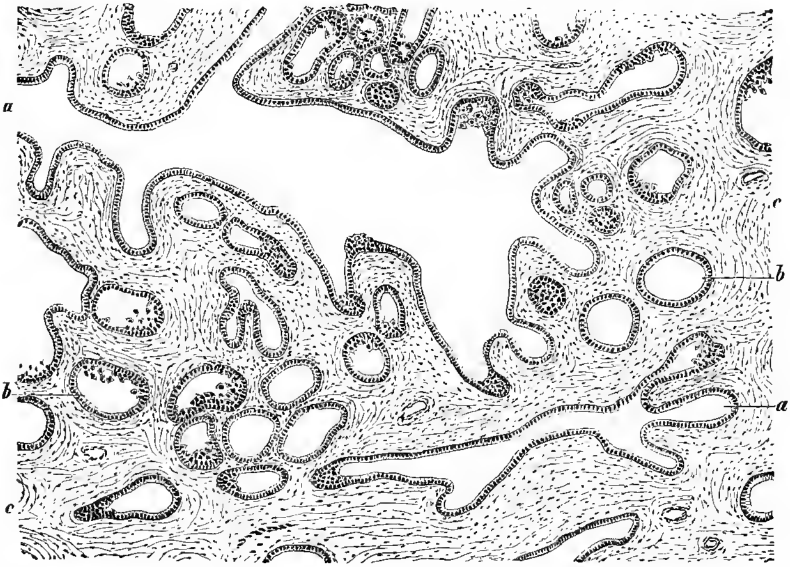

FIG. 541. TUBULAR ADENOMA OF THE BREAST.

(*Preparation hardened in alcohol, stained with alum-carmine, and mounted in Canada balsam:* × 30)

a longitudinal section of dilated and branching tubules
b cross-section of tubules
c stroma

In this group of tumours are included fibroma, myxoma, myxofibroma, sarcoma, and fibrosarcoma. When the proportion of glandular tissue they contain is still appreciable, they are described as adenofibroma, adenomyxoma, etc. All these tumours assume the form of well-defined nodes that are displaceable as regards the surrounding tissues; and even when the entire breast is involved the normal glandular tissue does not wholly degenerate, but is to some extent compressed and pushed aside by the growth. Such tumours are almost invariably unilateral, and many attain a very large size.

The surface is sometimes even, sometimes tuberous and lobulated: in the former case the cut surface is usually uniform in texture; in the latter the growth is more or less distinctly made up of nodules and nodes held together by tissue of a different structure.

The gross structure of these tumours varies with their origin and mode of growth. Those that are nodose and lobulated increase chiefly by the proliferation of the nucleated connective tissue (Fig. 542 *b*) which surrounds the ducts (*a*) and acini (periadenoid tissue), the fibrous stroma in general being at first but little altered. These might accordingly be termed periadenoid fibromata (Fig. 542). As the periadenoid connective tissue increases the glandular structures are thrust asunder, and the interacinous stroma is compressed into slender septa (*c*) between the fibrous nodes.

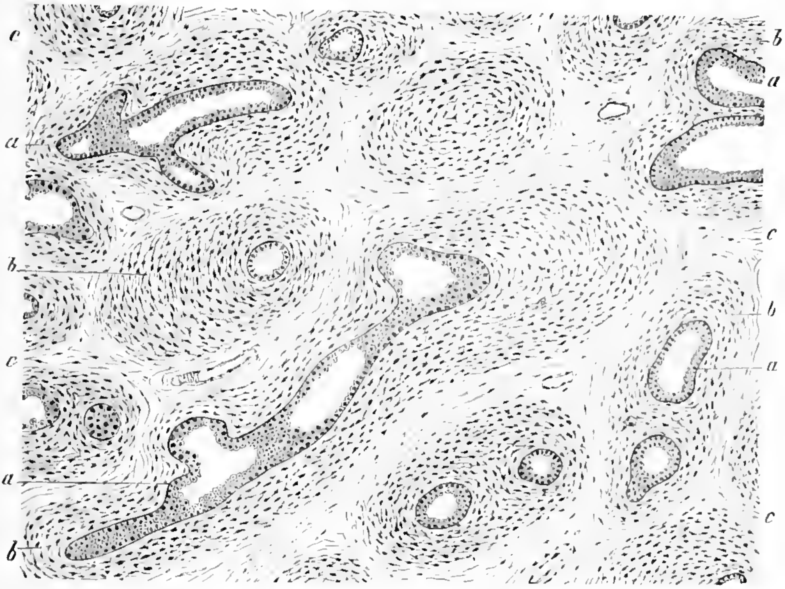

FIG. 542. PERIADENOID FIBROMA OF THE BREAST.

(*Preparation hardened in Müller's fluid and alcohol, stained with alum-carmine and eosin, and mounted in Canada balsam*: × 40)

a ducts *b* neoplastic periadenoid fibro-cellular tissue *c* loose fibrous tissue

Connective-tissue tumours with a uniform surface of section are produced by the neoplastic proliferation either of both these kinds of fibrous tissue or of the interacinous stroma alone; but a tumour derived from the stroma only sometimes possesses a lobulated structure.

The minute structure of the tumours in question is indicated by the names applied to them. It should however be remarked as regards the fibromata that soft and cellular varieties, as well as hard fibroid growths, are met with. Fibromyxoma is somewhat common, while simple myxomatous tissue rarely if ever constitutes a tumour of any great size.

Every variety of **sarcoma** is met with in the breast: round-celled and lympho-sarcomatous, spindle-celled and polymorphous-celled, giant-celled, alveolar, angio-sarcomatous, melano-sarcomatous (BILLROTH), and medullary striocellular types are all included among the recorded examples.

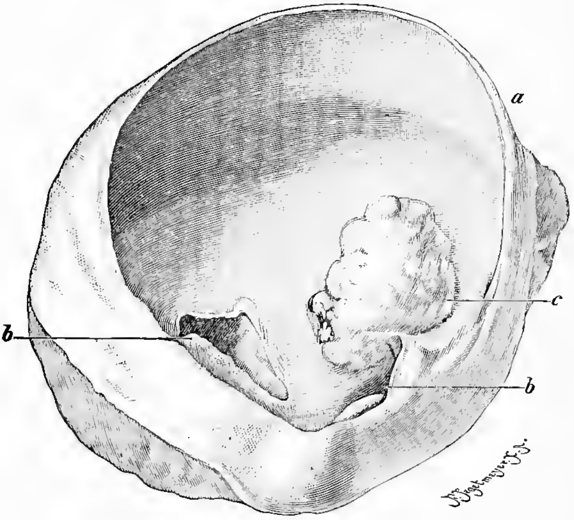

FIG. 543. PAPILLIFEROUS CYSTOMA OF THE BREAST.

(*The cyst was of twenty years' standing when the papillary excrescences developed: natural size*)

a large cyst *b* openings into smaller cysts *c* papillary excrescence

In all the connective-tissue tumours some glandular ducts and acini usually persist within the growth for a time; in the non-medullary forms indeed they not infrequently increase in size, their epithelium undergoing more or less abundant proliferation (Fig. 542 *a*). When this is the case the lactiferous ducts often assume, even to the unaided eye, the appearance of tortuous canals of the most various forms, while the cut surface appears to be traversed in all directions by a multitude of clefts running singly through it or branching into lateral fissures.

339. Many of the tumours of the breast that make their appearance in middle life and in old age are distinguished from

other mammary growths by the fact that they enclose cysts, and in view of this feature they may appropriately be grouped together under the head of **cystoma**. A cystoma may in the first place arise from the cystic dilatation of the neoplastic glandular tubules included in an adenoma or adenofibroma, and is then termed a **cystadenoma** or **cystic adenofibroma**. In other cases the formation of cysts is not preceded by the new-formation of tubules, and the cysts are then due to the dilatation of the existing ducts. In some instances it would seem as if the first step consisted in the distension with liquid of the ducts of a normal gland (Fig. 543), the neoplastic proliferation being a subsequent and secondary process. In others again the first stage is the neoplastic production of connective tissue, within which the cysts appear afterwards as secondary excavations: such growths are accordingly regarded as histioid tumours and described as examples of cysto-fibroma, cysto-sarcoma, or cysto-myxoma, as the case may be.

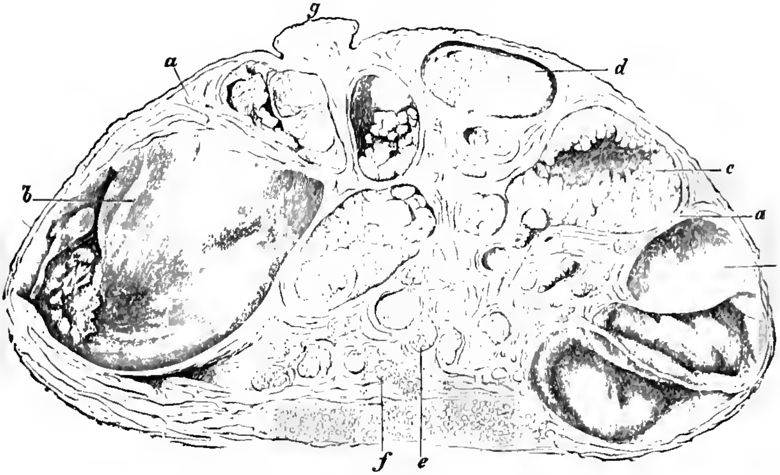

FIG. 544. PAPILLIFEROUS CYSTOMA OF THE BREAST.

(Two-thirds of the natural size)

a	stroma
b	smooth-walled cysts
c	cysts whose walls are beset with papillae
d	cysts entirely filled with papillae
e	small encapsuled papillary growths
f	adenomatous proliferation
g	nipple

The contents of the cysts consist mainly of thin viscid liquid; but some include spherules or 'pearls' of agglutinated and cornified epithelial cells, derived it is said from the squamous epidermoid epithelium with which some of the ducts are occasionally lined.

In cases of tubular adenoma, papillary excrescences are apt to make their appearance within the neoplastic tubules even when the tumour is of very small size, so that the neoplasm assumes from the outset the characters of a **papilliferous adenocystoma** or cystic papilloma. The papillomatous growths sometimes beset even the small and scarcely-dilated glandular tubules (Fig. 544 *e f*); thus at no time are any large cystic cavities formed that are simply filled with liquid and free from papillae, and the tumour accordingly consists of soft apparently-compact tissue (*e f*). More frequently however somewhat large cysts containing liquid are present, and these are only in part occupied by papillary growths (Fig. 544 *c*), or contain none at all (*b*). Cases are also met with in which the cyst-walls remain smooth for a long time (Fig. 543), and only begin to sprout into papillae under the influence of some morbid stimulus.

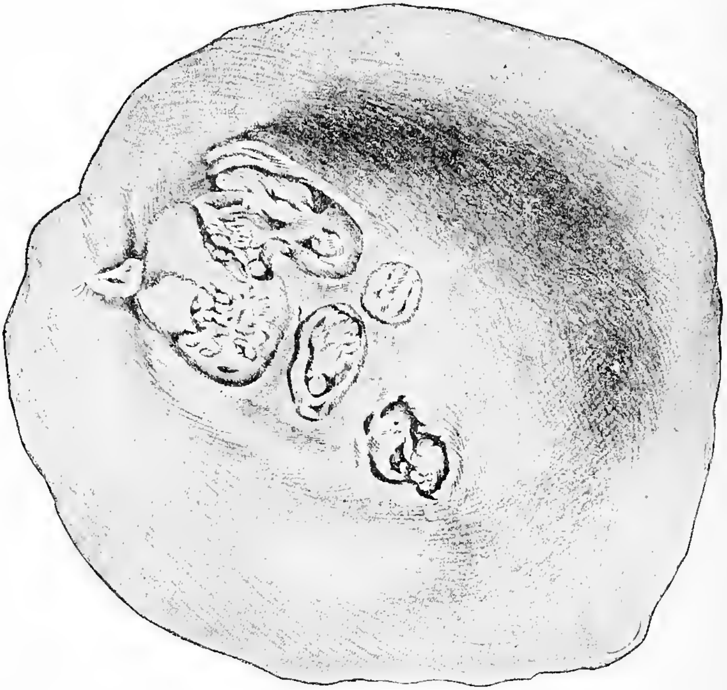

Fig. 545. Papilliferous cystoma of the breast whose papillae have broken through the cyst-walls and the skin.

(*Natural size*)

The intratubular papillae as they enlarge and multiply are checked in their growth by the pressure of the cyst-wall; but at times they break through it, and still enlarging ultimately penetrate the external skin (Fig. 545) and appear on the surface of the breast.

When in a papilliferous cystoma the production of neoplastic glandular tubules is very abundant, and the papillae are slender and delicate, the epithelial character of the growth becomes the predominant feature, and it is then described as an **epithelial cystoma.** In ordinary cases the epithelial investment of the tubules and papillae consists of a single layer of columnar or cubical cells; but occasionally the epithelium is more actively proliferous and overlies the slender papillae in several layers, the tumour thus assuming a medullary appearance and texture.

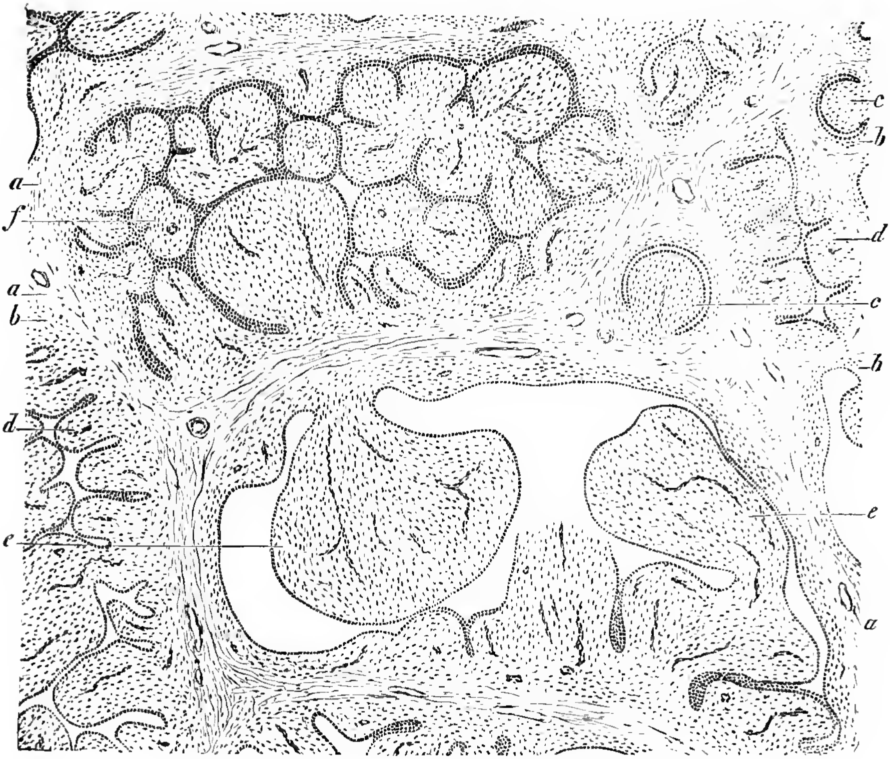

FIG. 546. INTRACANALICULAR FIBROMA OF THE BREAST.

(Preparation hardened in alcohol, stained with alum-carmine, and mounted in Canada balsam: × 25)

a	coarse intercanalicular fibrous tissue	*c d e*	longitudinal sections of intracanalicular polypous growths
b	periadenoid fibro-cellular tissue	*f*	cross-section of intracanalicular growth

The varieties met with in the case of ovarian cystoma thus find their analogues in the cystic tumours of the breast. The analogy extends to their clinical as well as to their histological characters, for when the epithelial proliferation and the production of papillae become exuberant, the mammary growths are apt to acquire a certain degree of malignancy, and pass without any abrupt transition into undoubted carcinoma. Accordingly it is not impossible for a proliferous mammary cystoma (Fig. 544) to give rise to secondary growths possessing the structure of carcinoma ; such a growth might fairly be described as a papillomatous **cysto-carcinoma**.

The periadenoid connective tissue of an adenoma or of a previously-healthy breast sometimes becomes proliferous, and growing in an irregular manner invaginates the glandular ducts and projects into their channels in the form of bulky papillary and polypous excrescences (Fig. 546 *c d e*). The ducts thus come to enclose voluminous fibrous, myxomatous, or sarcomatous growths that are overlaid only by an attenuated layer of epithelium (Fig. 546 *c d e f*). Tumours of this kind are termed respectively intracanalicular fibromata (Fig. 547), myxomata, and sarcomata.

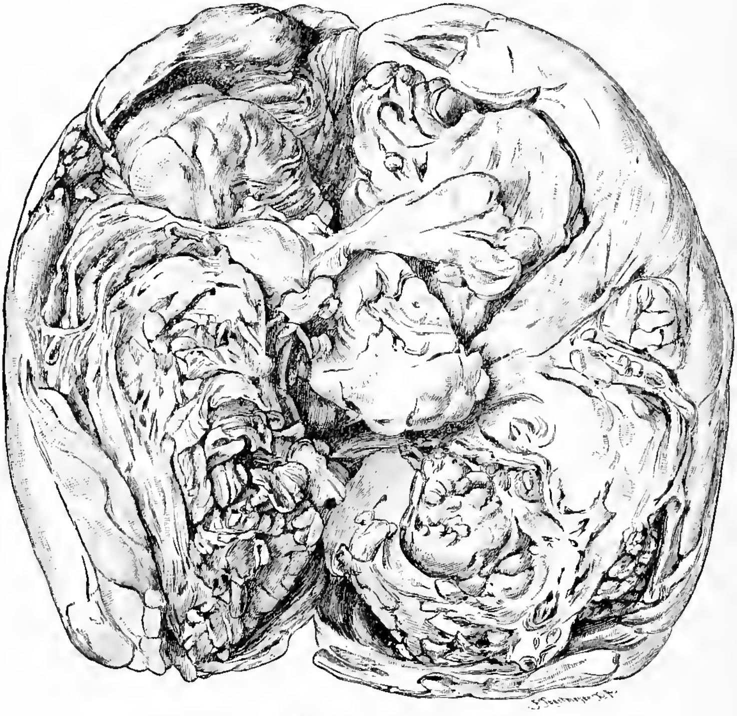

FIG. 547. INTRACANALICULAR LAMINATED FIBROMA (PAPILLOMATOUS CYSTOMA) OF THE BREAST.

(*The tumour is split open longitudinally : one-half the natural size*)

The unequal ingrowth of the connective tissue causes the glandular tubules to become stretched and deformed in the most various and extraordinary ways (Fig. 546 *d f*). If the ingrowing papillae attain any considerable size (Fig. 547) and subdivide more or less freely into branches, they are often compressed into rounded nodes, apparently enclosed in a cyst, and cleft by narrow fissures. On section the nodes exhibit a laminated structure due to the mutual compression of the intracystic papillae, and the growth has for this reason been termed **sarcoma phyllodes** (J. MÜLLER).

340. The commonest and the most malignant of mammary tumours is **carcinoma**, which as a rule appears between the thirtieth and the fifty-fifth year of age. In most cases one breast only is involved, but both occasionally become cancerous either simultaneously or in succession.

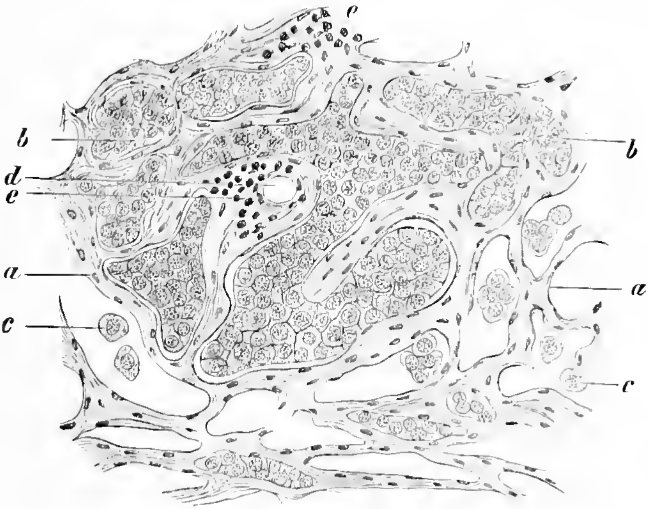

FIG. 548. CARCINOMA SIMPLEX MAMMAE.

(*Preparation hardened in alcohol, and stained with haematoxylin*: × 100)

a stroma
b cancerous loculi
c single cancer-cells
d blood-vessel
e cellular infiltration of the stroma

The growth always begins with the formation of more or less sharply-defined indurations or nodes which cannot be moved about in the gland-substance. As it continues to develope nodose or flattened tumours are produced, which are much indurated, and are often followed after a time by a certain amount of cicatricial contraction. The rate of growth may be rapid or slow, the flattened and contracting forms being generally slow-growing. According to BILLROTH the duration of a mammary cancer, up to its fatal termination by local extension and metastatic dissemination, varies from 6 months to 20 years.

According to the consistence of the individual nodes mammary cancers are distinguished as soft or hard, and according to the form and structure of the cell-nests as acinous or tubular; but one and the same tumour not infrequently exhibits different types of structure in different parts of its substance.

Medullary carcinoma is usually acinous in structure, and possesses a stroma which is often densely infiltrated with round-cells. In its interior degenerative changes usually make their appearance sooner or later; fatty degeneration is especially common, and often leads to softening and irruption of the growth through the skin, the opening being converted into a funnel-shaped or crateriform ulcer. From the floor of the ulcer rise bulky fungous masses of cancerous tissue with a granulomatous stroma.

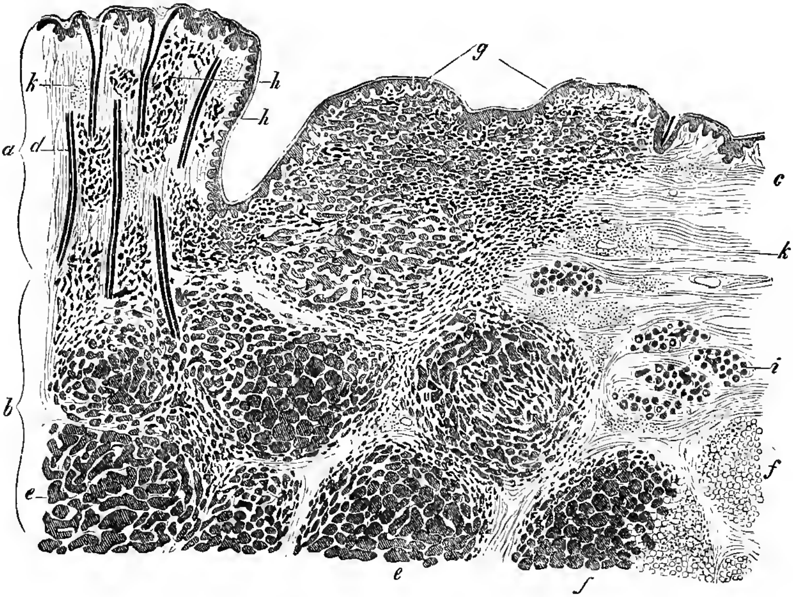

Fig. 549. Carcinoma of the breast.

(*Preparation hardened in alcohol, stained with alum-carmine, and mounted in Canada balsam:* × 6)

a	nipple
b	mammary tissue
c	skin
d	ducts
e	cancerous tissue
f	fat-lobule partially invaded by the cancer
g	cancerous skin
h	cancerous infiltration of the nipple
i	normal mammary lobules
k	cellular infiltration of the stroma

The metastatic growths that appear in the axillary glands possess a structure similar to that of the primary tumour.

Hard cancer, commonly known as **scirrhus**, forms either nodes that send out radiating processes and claws into the circumjacent tissue, or flattened indurations. Its cell-nests are sometimes irregular in shape (Fig. 548 and Fig. 549), sometimes rounded

(Fig. 549 *f*), or long and fusiform (Fig. 549 *g h*), and its fibrous stroma is somewhat abundant. The shape of the cell-nests (Fig. 549) depends mainly on the seat of the growth, varying with its position in the glandular parenchyma (*b e*), the adipose tissue (*f*), or the skin (*g*). Mammary cancers exhibit in the most typical way the tendency of the neoplasm to extend by infiltration. Once the papillary layer of the corium is pervaded by the cancerous infiltration, vesicles, crusts, and ragged fissures appear on the cutaneous surface, and the skin is at length penetrated and becomes ulcerous.

Sooner or later, in addition to the continuous infiltration of the immediately circumjacent tissues, a discontinuous mode of extension comes into play in parts a little more distant; then metastases are formed in remote organs; and at length nodular or diffuse infiltration and fibroid induration and thickening are apparent not only in the adjoining fat, muscle, skin, and lymph-glands, but often in the pleura, ribs, and sternum also. The skin over the front of the chest is sometimes beset with cancerous nodes and undergoes a diffuse form of induration (*cancer en cuirasse*). As the epithelial proliferation extends, the connective tissue generally becomes infiltrated with cells, the infiltration frequently anticipating the appearance of the cancerous cell-nests (Fig. 549 *k*). Presently the connective tissue increases in bulk and acquires a firm cicatricial texture. Retrogressive changes are always discoverable in this form of cancer also; they take the form chiefly of fatty degeneration and disintegration of the cancer-cells, some of which are thereupon re-absorbed and disappear.

Another slow-growing and very chronic form of carcinoma is specially characterised by the peculiarity that the cancer-tissue proper tends to waste and disappear, the stroma thereupon undergoing cicatricial contraction and induration: this form is accordingly described as contracting or cicatrising cancer, or **atrophic scirrhus.** When the growth lies near the nipple, the latter is retracted into the substance of the gland.

In rare cases of carcinoma simplex **gelatinous degeneration** of the epithelium (Fig. 550) supervenes, and the alveoli of the stroma become distended with a jelly-like substance (*carcinoma gelatinosum*). In such tumours there is usually no cicatricial induration or contraction, and the gelatinous substance is somewhat bulky; for these reasons the growth assumes a rounded or hemispherical shape.

Hyaline degeneration of the stroma, with subsequent **calcification,** is of very rare occurrence.

The forms of cancer that appear in the female breast are met with also in the male; but they are much less common, the cases not amounting to much more than three per cent. of the number of cases in women. Adenofibroma, sarcoma, etc., are very rare in the male breast.

A peculiar affection of the mammillary areola, resembling in some respects a chronic dermatitis, was described by Sir James Paget in 1874, and is now generally referred to as **Paget's disease.** It begins on one side with the formation of an erythematous and somewhat scaly patch, covered with small scabs, and this often becomes uneven, and it may be fissured and ulcerated. The border of the patch is raised and well-defined; and after it has existed with little change for some years the underlying tissues become cancerous. The ducts near their openings in the nipple are distended with proliferous epithelium, and it has been maintained (Thin) that the disease is primarily due to carcinoma starting in the ducts and so modifying their secretion as to render it irritating to the skin. In recent years several observers have supported the view that the affection is really of parasitic origin; certain rounded unicellular bodies which are found within the epidermal cells, and in the neoplastic epithelium of the subsequent cancer, being regarded as the exciting cause. These bodies are described as **Psorospermia**, and classed as animals belonging to the *Protozoa* (Darier, Hutchinson, and others). About the external genitals in men a somewhat similar affection of the skin is occasionally a precursor of cancerous infiltration; but the real nature of Paget's disease of the nipple is still a matter of dispute.

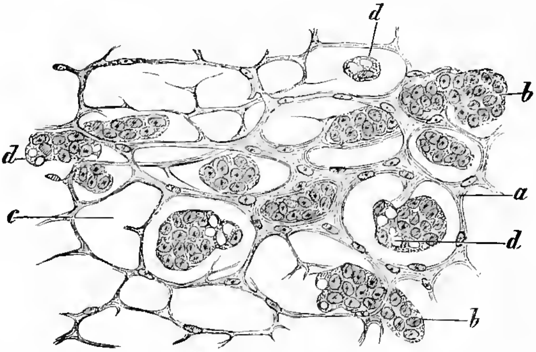

Fig. 550. Gelatinous carcinoma of the breast.
(Preparation hardened in alcohol, stained with haematoxylin, and mounted in Canada balsam: × 200)

a stroma
b cancerous loculi
c alveoli in the stroma
d cells containing spherules of colloid matter

Lipoma of the breast has no peculiarities distinguishing it from lipoma elsewhere, but it is not very common. Chondroma, osteoma, and angioma are very rare.

Echinococcus is practically the only **animal parasite** found in the breast, but it is seldom seen.

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SECTION XIV

THE EYE

(by Professor HAAB *of Zürich)*

CHAPTER CXI

MALFORMATIONS OF THE EYE

341. **Congenital defects** of the eye, other than those affecting the eye-lids, either involve the entire globe or are limited to certain parts of it. To the former category belong the anomalies known as anophthalmos, microphthalmos, and hydrophthalmos; to the latter, keratoglobus and coloboma of the iris and choroid.

Entire **absence** of the eye-ball is rare, rudimentary traces of it being present in most instances, so that the difference between anophthalmos and microphthalmos is really one of degree. **Microphthalmos** is the term applied to cases in which something bearing at least an external resemblance to the eye-ball is discoverable while in **anophthalmos** minute anatomical examination is usually necessary to make out any traces of an eye at all. Anophthalmos is met with either alone or associated with other structural anomalies, such as coloboma, hare-lip, absence of the interventricular septum of the heart, etc., and is usually bilateral. The fact that it is almost invariably the eye-ball alone that is wanting or rudimentary, while its appendages such as the lids, conjunctival sac, muscles, and nerves are frequently present, warrants the assumption that the eye had already attained a certain degree of development before its growth ceased and wasting began; and that, as a rule, the condition is essentially due to intra-uterine disease or atrophy of the globe. The anomaly is thus most probably referable to some disease of the foetus, as VON GRAEFE and H. MÜLLER have maintained in the case of microphthalmos.

Hydrophthalmos in early childhood is a result of increased ocular pressure (glaucoma) depending on some disorder of intra-uterine development, the nature of which is unknown. The eye, which in many instances even at birth shows signs of enlargement and cloudiness of the cornea, undergoes gradually-increasing glaucomatous distension: this is at first most apparent in its anterior segment, the line of junction between the enlarged cornea and the sclerotic becoming indistinct, and the anterior chamber being deepened. The enlargement is presently followed by cupping of the optic disc, attenuation of the sclerotic, softening of the vitreous, cataract, etc. (Art. 371).

Coloboma oculi is a result that usually involves both the iris and the choroid, though it is sometimes confined to one only of these structures. The cleft follows the lower part of the vertical meridian, its position corresponding to that of the original foetal cleft or choroidal fissure. In the iris it gives rise to an apparent enlargement of the pupil downwards, the aperture resembling a key-hole or an inverted gothic arch whose apex is at the attachment of the iris. The defect in the choroid is broad, rounded or oval in form, and extending to or even surrounding the optic disc; or it may be limited to a portion of the lower segment of the choroid. Over the defective region, instead of the choroid and retina, there is merely a delicate fibrous membrane containing scanty remnants of pigment and a few blood-vessels. The latter are partly retinal in origin, and partly derived from the posterior ciliary arteries and the vessels of the sclerotic. In some instances retinal elements are found within the limits of the coloboma, occasionally in considerable number. At the site of the coloboma, the globe sometimes bulges perceptibly (*ectasis*), in consequence of the thinness of its walls.

Among other malformations may be mentioned—congenital ptosis, or inability to raise the upper eye-lid; the presence of an epicanthus or fold of skin that passes like a bridge over the inner canthus of each eye; congenital absence of the iris (*irideremia*); persistence of the hyaloid artery; and persistence of the pupillary membrane. The latter, a somewhat frequent anomaly, assumes a variety of forms: the remnants of the membrane often appear as a group of small brownish or pale-coloured specks on the anterior capsule of the lens within the pupil; or as a group of very slender filaments crossing the pupil and attached to the iris about one millimetre from its edge; or again as somewhat compact membranous shreds. These all represent surviving fragments of the vascular envelope which in the foetus surrounds the entire lens, and which is supplied with blood by the hyaloid artery, a branch of the central artery of the retina passing through the vitreous from behind forwards: this artery itself disappears in the course of normal development. The venous efflux from the pupillary membrane takes place through the vessels of the iris.

Keratomegaly (*cornea globosa* or *keratoglobus*) is a rare congenital enlargement of the cornea unattended by any increase of intra-ocular pressure; the cornea remains perfectly transparent, and during life is sharply marked off from the sclerotic.

Another form of **ectasis** of the eye-ball is brought about by intra-uterine irido-choroiditis. In this form the enlarged cornea is from birth uniformly clouded, and the pupil is contracted owing to corneal adhesion of the iris.

Certain cases in which the orbit contains nothing but a cyst, with a small rudimentary globe-like appendage, may be regarded as extreme examples of ectasis.

Coloboma is generally regarded as the result of failure or imperfection in the closure of the choroidal fissure (MANZ). But in view of the facts that the cleft extends to the retina and its choroidal or pigmentary epithelium (representing the wall of the secondary optic vesicle), that the foetal iris is unfissured, that in the cleft region the retina may be present (Pause, Haab) though the choroid and the retinal pigment are absent, that the sclerotic shows signs of structural defect, and lastly that coloboma of the iris may be horizontal, it is not impossible that the initial disorder of development is not in the secondary optic vesicle but in the inner or cephalic plate of the primary vesicle. As a matter of fact, Deutschmann has shown that intra-uterine sclero-choroiditis is capable of giving rise to coloboma of the choroid and iris.

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CHAPTER CXII

DEGENERATION AND ATROPHY OF THE EYE

342. Primary **atrophy** of the **conjunctiva** is a rare affection; but a textural or so-called essential degeneration known as **xerophthalmia** is met with, in which the conjunctiva gradually undergoes a sort of cicatricial contraction. This in time leads to the entire obliteration of the upper and lower conjunctival sacs, the lids becoming firmly adherent to the eye-ball (symblepharon). The resulting changes in the cornea are due mainly to drying up of the epithelium and to consecutive inflammation of the conjunctiva.

Secondary atrophy of the conjunctiva is common enough as a consequence of penetrating lesions like burning and cauterisation, and of diphtheritic inflammation, trachoma, etc.; it is manifested by cicatricial contraction and obliteration of the conjunctival sac. **Pemphigus** of the conjunctiva gives rise to appearances resembling xerophthalmia. It may indeed be that in the latter condition pemphigus is always a factor (SCHWEIGGER, ALBRAND).

Xerosis is a more superficial affection, in which the epithelium of the ocular conjunctiva between the lids appears to lose its moisture, becoming lustreless and spotted with small white fatty-looking scales, which sometimes appear also upon the adjacent cornea. The disease may disappear of itself, or lead to inflammation: it occurs usually in poorly-nourished patients. The fat-like masses deposited on the scleral conjunctiva consist of successive layers of epithelial scales that have undergone cornification and fatty degeneration; in addition to minute globules of free fat they generally contain numerous bacteria (bacilli and micrococci), which by some have been regarded as the exciting cause of the affection.

The part of the ocular conjunctiva lying within the palpebral fissure to the right and left of the cornea is often overlaid by a slightly-elevated yellow patch or **pinguecula**. According to FUCHS, it is due to hyaline degeneration of the conjunctiva and subconjunctival tissue, accompanied by the production of numerous large elastic fibres which themselves undergo hyaline change.

Amyloid degeneration of the conjunctiva is rarely observed: it may appear as a primary change in healthy persons with perfectly normal mucous membranes, but more frequently it is secondary to trachoma. In both cases the affection runs a chronic course without any marked symptoms of inflammation, and it leads to great enlargement and thickening of the lids, owing to the formation in them of diffuse or nodular deposits of a yellowish-white or yellowish-red waxy colour, that are sometimes dry and brittle, sometimes more like bacon-rind or stiff jelly. The degeneration appears on one side or both, and may affect all four lids or be confined to one. Closely allied with it is the still less common **hyaline degeneration** of the conjunctiva, which in eyes that are otherwise entirely sound gives rise in like manner to the formation of nodular or lobulate swellings, or to irregular deposits.

The relation of hyaline to amyloid degeneration is discussed in the volume on General Pathological Anatomy. Transformation of the products of hyaline degeneration into amyloid substance has been observed in the case of the conjunctiva by some (RÄHLMANN, PORIWAËW), but others have failed to verify the observation (VOSSIUS, KAMOCKI). VOSSIUS found that the fibres and vessels of the conjunctiva were the usual site of the hyaline as well as of the amyloid deposit, and maintained that the connective-tissue cells take no active part in the formation of either of the substances. RÄHLMANN, LEBER, and others, have made out that the tissue-cells participate in the production of the amyloid substance, and RÄHLMANN asserts the like as regards the formation of the hyaline substance.

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343. **Degeneration** of the epithelium of the **cornea** generally takes place whenever the cornea is for any considerable time deprived of the protection of the eye-lids. This is especially the case in extreme exophthalmos, due for instance to intra-orbital tumour, in eversion of the lids (ectropion), and in corneal staphyloma of large size (Art. 344). In all these affections the epithelium thus exposed to the air becomes dry, and as it is not detached it renders the surface lustreless, rough, and cloudy; and the cornea at length becomes studded with whitish cicatrices that often enclose spots of yellow. These latter are due to deposits of hyaline substance (FUCHS).

Linear (VON GRAEFE) or **zonular opacity** (VON ARLT) of the cornea is a somewhat infrequent affection of more advanced life, commonly present in both eyes though not always simultaneously, and usually unaccompanied by inflammation. It assumes the form of a sharply-defined turbid zone, from 2 to 4 millimetres wide, which runs transversely across the lower part of the cornea. Within this zone the surface of the cornea is dotted with minute greyish or brownish spots, and is thereby rendered remarkably opaque. The epithelium is greatly thickened in places, and beneath it hyaline concretions (FUCHS) are deposited, together with thin layers of a hard substance containing carbonate and phosphate of calcium and magnesium in the form of fine granules and crystals.

Zonular opacity of the cornea is met with (1) in eyes undergoing contraction in consequence of inflammation of the iris and ciliary body (irido-cyclitis), (2) in eyes affected by chronic glaucoma (Art. 371), (3) much less frequently in senile eyes that are otherwise normal. In the last case the colour of the opacity inclines more to greyish-yellow or greyish-brown (VON ARLT).

In advanced age the margin of the corneal conjunctiva is often the seat of a finely-granular deposit of hyaline substance beneath Bowman's membrane (FUCHS), forming the so-called **arcus senilis** (*gerontoxon*). This light-grey arc is concentric with the upper and lower parts of the circumference of the cornea, from which it is separated by a narrow strip of transparent tissue. On the nasal and temporal sides the zone is usually narrower or absent, but it sometimes forms a complete circle or *annulus senilis*.

When the corneal tissue loses its power of resistance and so yields to the intra-ocular pressure, its middle part is apt to bulge forwards and gradually to assume the form of a cone, with a rounded apex occupying approximately the centre of the cornea, a condition known as **keratoconus.** In advanced cases of some standing the summit of the prominence becomes thinned out and somewhat greyish-white in colour. The affection is however comparatively infrequent. It is highly probable that a certain abnormal thinness of the central portion of the cornea is the cause or at least a contributory condition of the development of keratoconus.

The *substantia propria* of the cornea occasionally becomes yielding and inelastic as a result of chronic inflammation accompanied by the formation of numerous blood-vessels (*pannus*), and giving way in like manner it protrudes in a conical form (*keratectasia ex panno*).

344. **Staphyloma of the cornea** is a hemispherical protrusion of part or the whole of the structure; it is usually of a bluish-grey colour and looks not unlike a white grape. It is filled with aqueous humour, and consists entirely or partially of cicatricial tissue containing a variable amount of pigment. The wall of the staphyloma may be thick throughout (2 to 3 millimetres or more), or thin and in some parts so weak as to give way altogether by rupture.

Staphyloma of the cornea is liable to follow upon extensive ulceration with perforation of the cornea. When the iris is forced into the perforation (*prolapsus iridis*), and serves in part to fill up the gap as the ulcer heals, the resulting cicatrix is weaker than the rest of the cornea. The traction on the iris gives rise to increased (glaucomatous) pressure within the eye, and this steadily forces out the yielding cicatrix. When the ulceration is small in extent the result is in some cases merely a cicatricial ectasis with anterior adhesion of the iris (*synechia*). If the loss of substance is considerable and exposes a large portion of the iris (over 4 millimetres), partial staphyloma is usually produced; while destruction of the greater part or the whole of the cornea naturally gives rise to total anterior staphyloma.

This last condition is apt to bring about what is called **intercalary staphyloma**, a local or general protrusion of the anterior zone of the sclerotic, over a strip about 3 millimetres wide immediately adjoining the corneal limbus, the protrusion being more or less completely annular and made up of contiguous slaty-grey or dark-blue prominences.

Scleral staphyloma is generally dependent on atrophic attenuation of the sclerotic, and takes the form of an ectatic protrusion either in the anterior segment, at the equator, or at the posterior pole of the globe.

Ciliary staphyloma is somewhat posterior to the site of intercalary staphyloma, its position corresponding to that of the ciliary body. It is sometimes due to inflammation of the sclerotic (scleritis). As the inflammation subsides the sclerotic bulges out as an annular prominence (VON ARLT) about the anterior scleral zone, and in consequence of the thinness of its wall acquires a slaty-grey colour. This termination of scleritis is however a very rare one. Scleral staphyloma of the ciliary or equatorial region is much more commonly induced by chronic sclero-choroiditis, which usually begins first in the choroid as choroiditis or irido-choroiditis, and then gradually extends to the sclerotic. In the course of the affection the intra-ocular pressure is generally increased, and this is the chief agent in augmenting the protrusion. Both the ciliary and the equatorial forms of staphyloma are usually multiple, the latter being often of considerable extent. Anatomical investigation shows that in the later stages the stretched and attenuated sclerotic is in general closely adherent to the atrophic choroid, and that the retina over the staphylomatous region is also much atrophied and firmly united with the choroid; less frequently the retina is detached and bridges over the concave aspect of the ectatic portion.

Staphyloma of the posterior pole of the eye-ball is now and then caused by sclero-choroiditis of this region. As a rule however the signs of antecedent inflammation are not very distinct. The pathogenesis of Scarpa's posterior staphyloma accompanying extreme myopia is not yet completely determined (Art. 349).

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345. **Cataract** is an opacity of the lens due to changes in its own tissue or in its capsule and capsular epithelium. The opacity is in some cases grey and in others white; it may occur at any time of life; and is either idiopathic or secondary to other ocular affections such as chronic choroiditis.

In the new-born infant all the fibres of the lens are soft and pliant, but early in youth a process of hardening begins in the centre of the lens, and steadily increases with advancing age. In very old persons only the cortical stratum of the lens remains soft, while the central nucleus is quite hard and is somewhat yellowish in colour. The hardening is effected by a keratinoid metamorphosis (which however is not due to the formation of keratin) and by coalescence of the crystalline fibres, which lose their nuclei, become flattened, and blend into a homogeneous mass with little or no trace of concentric arrangement. In senile cataract it is the part of the lens which remains soft, in other words the cortical layer, that becomes most opaque.

The opacity of the lens in cataract is at the outset due to the presence within its fibres of vacuoles and small droplets of liquid, to separation of its fibres, and to the appearance in the interstices thus formed of homogeneous spherules. The separation of the fibres first takes place in the equatorial region. Presently the majority of the fibres appear granular and turbid, then become transversely striated, and finally break down into granular detritus and drops of myelin. As the lens swells up while these changes are taking place, it is evident that the process of degeneration is attended by an increased imbibition of liquid into its substance.

As the crystalline fibres disintegrate the lens is by degrees converted into a pulpy and partly calcareous mass of detritus consisting of fat-granules, drops of myelin, cholesterin-crystals, and broken-down fibres. When much water is present in the mass it becomes liquid and milky-looking, and the still firm central nucleus sometimes sinks through it (Morgagni's cataract). If, as is usually the case, the detritus loses some of its component water and so becomes inspissated, the lens shrinks and becomes smaller, and at the same time a capsular cataract begins to form. This is the stage of 'over-maturity.' The firm nucleus that has itself escaped disintegration undergoes a change in its chemical composition while the cataract is in course of formation, the change consisting essentially in an increase of its cholesterin (ZEHENDER, MATTHIESSEN and JACOBSEN, MICHEL and WAGNER).

The capsule of the lens does not exhibit any noticeable loss of transparency during the formation of the cataract; but it sometimes becomes considerably thicker on both the anterior and the posterior surface, and acquires a faintly-striated appearance. On its inner surface, moreover, transparent deposits in the shape of flattened, rounded, or conical elevations are liable to be formed, the matter of which they are composed being very similar in its properties to the substance of the capsule itself: it is probably either of cuticular origin, or a derivative of the capsular epithelium. In support of the latter probability may be adduced the fact that, according to BECKER, the nuclei of the capsular epithelium undergo in capsular cataract a peculiar transformation, being converted into a homogeneous reddish-brown highly-refractive substance, which, as the cells dissolve, forms grumous deposits on the inner surface of the anterior capsule.

While the cataract is forming the capsular epithelium occasionally undergoes dropsical degeneration, its cells swelling up by imbibition, and enlarging sometimes to an enormous size (vesicular cells of BECKER). The exterior fibres of the equatorial zone of the lens may likewise be converted into vesicular cells, and then as their nuclei disintegrate undergo liquefaction. The change is chiefly due to imbibition of liquid, and it occurs not only in cases of simple cataract, but also in inflammatory conditions such as irido-choroiditis, and after operations for the extraction of cataract.

The epithelium of the anterior capsule is prone to become proliferous both in uncomplicated cataract and after wounds of the capsule and inflammations about it. The cells as they increase in size and number force their way between the unaltered epithelial cells, and so raise them from the capsule. They also send out processes that unite with similar processes from other cells, and in this way form a cellular network in whose meshes the remaining epithelial cells are enclosed. A homogeneous intermediate substance by and by makes its appearance between the epithelial cells, and the structure, originally consisting of nothing but cells, is thus transformed into stratified tissue not unlike that of the cornea, and usually separated to a varying extent from the crystalline fibres by a stratum of normal epithelium. After a time retrogressive changes generally take place in this semi-fibroid product of the capsular epithelium, and result in the formation of highly-refractive flakes, plates of cholesterin, and chalky deposits. The outcome of all is that the capsule becomes white and opaque.

These changes in the capsule and its epithelium together constitute the condition known as **capsular cataract**, which is liable to accompany both congenital and acquired cataract of the lens, whether senile or secondary to other ocular affections.

In addition to the overgrowth of the anterior capsular epithelium, epithelioid cells not infrequently make their appearance on the inner surface of the posterior capsule, and there form continuous or discrete cellular deposits (the pseudo-epithelium of the posterior capsule of BECKER). The cells are sometimes round, sometimes oval, and sometimes irregular or stellate: they now and then form on the posterior capsule a texture very similar to that just described as investing the anterior capsule, and some are occasionally converted into vesicular cells.

As the posterior capsule normally possesses no epithelium of its own, it is very probable that these cells are derived from the anterior capsule. It is not unlikely (BECKER) that the loosening and separation of the ends of the crystalline fibres allow the epithelium of the anterior capsule to grow backwards round the equator of the lens.

In the course of inflammations affecting the parts about the lens, pus-corpuscles at times break through its capsule and force their way into its interior. But so long as the capsule remains intact, pus is never formed within the lens itself.

Cataracts have received special names according to their position, extent, and characters, and according to their mode of origin, and the time of life at which they appear. Thus a cataract is lenticular when only the substance of the lens is affected, and capsular when only the capsule and its epithelium are involved. By such terms as total, partial, and central or axial cataract, or *cataracta lactea*, *nigra*, *fluida*, etc., the seat, extent, and appearance of the morbid changes are indicated; while the terms senile, juvenile, congenital, primary, secondary, etc., have reference to the time of appearance or to the pathogenesis of the cataract. Certain special points relating to some of the principal varieties require to be noted.

Congenital lenticular cataract, other than the variety with a hard core, concerning whose minute structure little is known, is always soft throughout at the time of birth. In these cases, according to BECKER, an abnormal quantity of liquid is taken up by the lens and accumulates between the capsule and the crystalline fibres. The disintegration of the fibres that subsequently takes place is sometimes so complete that at length nothing remains but a little bag or vesicle filled with liquid detritus.

Lamellar cataract (*cataracta zonularis*) is either fully formed at birth, or developes in the early years of childhood. The peculiarity of this form is that only a single stratum of the lens becomes opaque. The central nucleus remains transparent, and is enveloped by the opaque layer as by a shell: as the latter varies greatly in size, the width of the transparent cortical zone varies also in different cases. Traces of a second opaque stratum can sometimes be detected outside the first about the periphery of the lens. The second layer is concentric with the first, and in rare instances appears to be complete: cases have indeed been recorded in which portions of a third layer were present. That this variety of cataract is related to rickets is now beyond question (HORNER).

Microscopical examination of the affected layer reveals that its opacity is due to the presence between the crystalline fibres of slender rounded or spindle-shaped spaces or clefts filled with finely-granular matter and drops of myelin. The central nucleus also is traversed by minute fissures, although in much smaller number, while the cortical layer is normal.

Another form that is either congenital or acquired in early youth is termed **pyramidal cataract** (*cataracta polaris anterior*). It is confined to the capsule about the anterior pole of the lens, sharply-defined and often conical in form with its apex towards the cornea, and but slightly prominent; its diameter at the base varies from 0·5 to 2·0 millimetres. The opacity is remarkably white, and the altered capsule is in general slightly corrugated. The cataract is due to ulcerous perforation of the cornea in early life, such as is apt to follow upon infantile blennorrhoea. When the aqueous humour escapes, the lens comes into contact with the cornea at the point of perforation, its capsule adheres to the cornea, and the capsular epithelium then becomes proliferous. Should the anterior chamber be re-formed, the lens recedes from the cornea, and the capsule is drawn out into a cone or pyramid with its apex at the point of adhesion.

Posterior polar cataract is the name given to an opacity that begins at the posterior pole of the lens as a small circumscribed spot; this often remains stationary for a long time, but ultimately extends in the form of radiating streaks within the cortical layer. Upon what the primary opacity depends is not known; as the process advances the crystalline fibres are forced asunder, and spherules of coagulated matter appear between them (LANDOLT, BECKER). At the same time the epithelium of the capsule undergoes proliferation, which leads to a deposit of cells on its posterior surface (pseudo-epithelium). This affection supervenes on diseases of the choroid, vitreous, and retina (especially in connexion with retinitis pigmentosa), and is apt to follow upon detachment of the retina, the growth of intra-ocular tumours, and cyclitis.

FUCHS and others distinguish between true posterior polar cataract (usually congenital) and the posterior cortical cataract described in the text. The two conditions are said to differ in their causation, structure, and pathology, and to admit of differential diagnosis during life (FUCHS: *Lehrbuch. d. Augenheilkunde* 5th edition **1894**).

Traumatic cataract is usually the result of a wound of the capsule of the lens. If the capsule is torn, the contiguous liquid makes its way into the substance of the lens, and causes swelling and turbidity of its fibres. According to SCHLOESSER, the fibres first become vacuolated; then the posterior cortical stratum becomes swollen, and the anterior stellate rays appear beset with granular matter, revealing the existence of a system of canaliculi around the central nucleus. In young persons the greater part of the substance of the lens is often extruded through the rent, and gradually dissolves in the aqueous humour. Even when but a small portion of the crystalline substance escapes, the part that remains within the capsule generally becomes altogether opaque. It is only in rare cases that wounds of the lens in man heal without the formation of a cataract. Reproduction of the substance of the lens or its capsule takes place only to a very limited extent. Renewal of the capsule, when it does occur, is effected by the proliferation of the capsular epithelium. Violent concussion has been known in certain cases to induce turbidity or opacity of the lens.

Senile cataract is due to opacity of the cortical layers of the lens, the firm sclerosed nucleus remaining more or less transparent. The opacity usually begins in the equatorial parts of the cortical stratum, and spreads through both its anterior and posterior layers. The first stage of the process consists in separation of the fibrous layers or coats of the lens, and is followed by molecular turbidity and disintegration of the fibres themselves.

The variety of cataract that appears in the course of **diabetes** presents no features peculiar to the disease. The textural changes in the lens or capsule are the same as those observed in non-diabetic cataract occurring at the corresponding period of life.

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BESELIN: *A. f. Augenheilk.* XVIII **1887**

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346. Atrophy and degeneration of the uveal tract (choroid, ciliary body, and iris) are met with chiefly in old age, as the result of inflammation or glaucoma. The anterior segment of the **choroid** included between the equator of the eye-ball and the ciliary body, and the parts about the optic disc, are the portions most liable to **senile atrophy**. In these regions, after the sixtieth year, great attenuation of the membrane and obliteration of its capillary layer not infrequently take place (KUHNT).

The structures known as **colloid nodules** of the hyaline membrane (colloid degeneration of the choroid), first carefully studied by DONDERS and H. MÜLLER, are of frequent occurrence in the eyes of the aged, and may often be recognised with the ophthalmoscope as small bright points scattered over the equatorial region of the choroid or around the optic nerve. They are seated on the innermost layer of the choroid (*lamina vitrea*), and project from it into the pigmentary layer, which they to some extent push aside. They are bulbous in form and concentrically-laminated in structure, staining faintly with haematoxylin and carmine, and resembling the analogous senile excrescences found on Descemet's membrane and on the inner surface of the anterior capsule. These little nodules were formerly regarded as outgrowths from the hyaline membrane, but recent researches render it more probable that they are derived from the pigmentary epithelium. Exactly similar structures are often produced as a result of chorio-retinitis and retinitis pigmentosa. According to VON RECKLINGHAUSEN the excrescences are composed of hyaline substance.

Inflammation is followed by well-marked atrophy of the **iris**, chiefly in cases where the iris becomes adherent to the cornea (anterior synechia, *leucoma adhaerens*) and so is subjected to constant traction. The wasting is manifested by loss of the pigment, and by attenuation and fibroid degeneration of the iris-tissue. The coats of the blood-vessels undergo hyaline degeneration and thickening, their channels being thereby obstructed or sometimes even completely occluded (ULRICH). A similar degeneration of the iris takes place in chronic glaucoma.

The stretching to which the choroid is subjected in staphyloma leads to precisely similar atrophic changes in this membrane, which as a rule becomes firmly adherent to the distended sclerotic.

Inflammatory processes affecting the choroid leave behind them cicatricial patches, diffuse or circumscribed, the tissue of which is fibrous, the vessels obliterated, and the nerves destroyed, while adhesions of the choroid to the adjoining parts of the retina and sclerotic are set up at the same time.

After severe purulent inflammation of the venous (vorticose) layer, **ossification** may gradually and after the lapse of a considerable time take place within the region originally occupied by the exudation, especially towards the inner aspect of the choroid.

Senile changes in the **ciliary body** affect principally its non-corrugated portion, and take the form (KUHNT) of thickening and abnormal vascularity of the reticular layer (Brücke's muscle), the production of sprout-like excrescences projecting into the vitreous, and the development of cysts due to detachment of the pars ciliaris of the retina from the pigmentary layer. These cysts sometimes measure from 6 to 7 millimetres across. The pigmentary layer itself is also not infrequently detached, and takes part in the formation of the cysts. The part of the ciliary body within which the cysts are situated exhibits atrophic changes distributed over certain of its vascular territories (KUHNT).

In extreme myopia and after various inflammatory conditions the ciliary body is liable to undergo atrophic changes of the same kind.

Degeneration of the **vitreous body** is common after inflammation of the choroid, and especially after such as affects the ciliary body. It is also apt to supervene in extreme myopia.

In its normal condition the vitreous body is a clear gelatinous spheroid, containing a few polymorphous cells; but in the course of some of the affections just described it is very apt to become liquid and diffluent (*synchysis*). At the same time foreign elements appear within it in the shape of minute specks, filaments, and membranous shreds, which are probably derived for the most part from immigrant lymph-cells, residues of extravasated blood, and the like. Cholesterin-crystals are often present, and (according to PONCET) needles of tyrosin and phosphatic particles also (*synchysis scintillans*). Not infrequently the posterior part of the vitreous body is separated from the retina by a serous effusion (IWANOFF's detachment of the vitreous). The degenerate vitreous often becomes shrunken (Art. 347).

Degeneration and shrivelling of the entire eye-ball, such as sometimes follow penetrating wounds and certain inflammations (especially those of the uveal tract), constitute the condition known as **atrophy of the globe** (*phthisis bulbi*).

Shrinking of the anterior segment of the eye (anterior atrophy) is commonly due to chronic inflammation of the ciliary body (cyclitis), the adjacent parts of the iris and choroid being implicated to a greater or less extent. At a certain stage of the process the eye becomes abnormally soft, and the pressure of the rectus muscles accordingly tends to make the globe assume the form of a four-sided prism.

Atrophy of the entire eye, producing analogous changes, generally follows purulent inflammation of the choroid, with suppuration of the vitreous. This form of inflammation may be set up by infection either from without (wounds, foreign bodies, corneal ulcers), or from within (pyaemic metastasis, meningitis). In the course of the process the eye slowly shrinks to a mere fraction of its normal bulk.

On examination the wasted and shrunken eye-ball usually exhibits marked thickening of the sclerotic, and atrophy of the choroid and retina, the latter being often detached or absent in consequence of the antecedent suppuration. The vitreous is in general shrivelled up into a small fibrous remnant. In long-standing cases the vitreous is not infrequently replaced by an osseous deposit, the entire posterior segment of the globe being filled up with spongy bone. Between the osseous trabeculae are vessels and abundant connective tissue, occasionally enclosing calcareous concretions.

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MÜLLER, H.: *ibidem* II **1856**
VON RECKLINGHAUSEN: *Handb. d. allg. Pathologie* Stuttgart **1883**

References on Atrophy of the Uveal Tract (Choroid, Iris, and Ciliary Body).

KUHNT: *Klin. Monatsbl.* **1881**
ULRICH: *A. f. Ophth.* XXVIII **1882**
VON WECKER: *Graefe u. Saemisch's Handb.* IV Leipzig **1876**

347. Degenerations of the **retina** are incidental to old age, when it becomes less transparent, while its external and internal limiting membranes are thickened, and the walls of its blood-vessels become sclerotic and infiltrated with oil-globules or calcareous granules (LEBER). In aged persons cystic degeneration also is common in the anterior segment of the retina; and a similar change involving the whole of the membrane is apt to supervene in cases of detachment of the retina, of traumatic injury, and of glaucoma. The development of the cysts begins with the formation of rounded lacunae which (according to MERKEL) are generally situated in the outer and less frequently in the inner nuclear layer: according to KUHNT, cysts may also appear in the molecular and ganglionic layers, and in the layer of nerve-fibres. In this way are gradually formed rows of contiguous intercommunicating cavities, separated from one another by arcades of thick radial fibres, and containing liquid. As the lacunae coalesce they give rise to cysts which sometimes measure several millimetres in diameter.

Atrophy and degeneration of the retina also follow upon chronic retinitis (Art. 369), haemorrhage, detachment of the retina, chronic choroiditis, and extreme morbid alterations in the retinal vessels, such as result from embolic or thrombotic occlusion. The nervous elements suffer most in retinal atrophy, the supporting structures and the pigmentary epithelium not infrequently undergoing hyperplastic proliferation. The rods and cones, before they disintegrate, often swell up and assume a clavate or pyriform shape. At times (especially in cases of detachment) they become elongated to twice or thrice their normal length, change their form, split at their ends into a number of filaments, become globular or flask-shaped, or are converted into highly-refractive fibrillar columns (LEBER). The nerve-cells of the outer and inner nuclear layers, as well as the ganglion-cells, occasionally perish by fatty and colloid degeneration. Extensive haemorrhage and severe inflammation speedily render them necrotic. Cases occur in which all the nervous elements of the retina are destroyed, nothing remaining of the membrane but a film of connective tissue.

The **pigmentary epithelium** of the retina seldom if ever remains intact when the choroid and retina are diseased; but it is also liable to undergo various independent morbid changes. The cells usually lose their regularity of form and pigmentation, some being deprived of pigment altogether and others becoming more deeply pigmented. It has already been stated that the so-called colloid nodules of the vitreous lamina are now regarded as derived from the cells of the pigmentary epithelium.

Especially important, and by no means infrequent, is the degeneration of the retina and its pigmentary epithelium about the yellow spot which is met with in old age, and in cases of traumatic injury such as crushing or bruising of the eye-ball, wounds from foreign bodies in the vitreous, etc.

Detachment of the retina (*solutio* or *amotio retinae*), the separation of the retina from the choroid, or more correctly speaking from the pigmentary epithelium, by a liquid effusion which is generally of a serous kind, is in nearly all cases a secondary process due to causes extrinsic to the retina; in a few instances however it is brought about by intrinsic changes such as shrinking of the membrane itself. The condition is usually induced by—(1) Traction on the retina, tearing the membrane from the underlying structures, and exerted by the vitreous when it is diseased and contracts (LEBER, NORDENSON) or when it loses a portion of its substance through a wound: such shrinking of the vitreous is very apt to follow upon certain inflammations of the uveal tract and in cases of extreme myopia; it constitutes the commonest cause of detachment of the retina. (2) Separation of the retina from its substructures by underlying growths such as *Cysticercus* and tumours of the choroid (sarcoma, tuberculous nodes, etc.), the membrane being either in contact with the growth or separated from it by a liquid effusion. In somewhat rare instances the detachment is due to subretinal inflammatory exudation, oedematous transudation, or haemorrhagic effusion, spontaneous or traumatic.

That contraction unaccompanied by fibroid degeneration of the vitreous is capable of inducing detachment of the retina has been demonstrated by the experiments of LEBER. A few days after he had introduced aseptic foreign bodies into the vitreous chamber of a rabbit, he observed that the retina became detached over a wide area. This result could have been produced only by the traction exerted on the retina by the vitreous as it thickened and contracted.

According to LEBER, detachment is facilitated by the rents produced in the retina at the points of maximum traction. Through these rents, which in a certain proportion of cases of detached retina can be detected with the ophthalmoscope, and which were invariably present in the rabbit's eyes experimented on, liquid from the vitreous chamber makes its way behind the retina, and rapidly enlarges the area of detachment.

NORDENSON'S investigations confirm those of LEBER, and show that chronic contraction of the vitreous, without any loss of its transparency, is accompanied by the development in it of fibrillated flocculi. The space between the vitreous and the retina, left vacant as the former shrinks, becomes filled with serous liquid, especially in the posterior region of the globe; while the anterior portion of the retina, which is more intimately connected with the vitreous, is apt to be dragged inwards. The shrinkage of the vitreous is ultimately dependent on chronic choroiditis.

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LIEBREICH: *A. f. Ophth.* v **1859**
NETTLESHIP: *St Thomas's Hosp. Reports* VIII London **1878**
NORDENSON: *Die Netzhautablösung* Wiesbaden **1887**
STELLING: *A. f. Augenheilk.* XII **1883**

348. Atrophy of the **optic nerve** is a somewhat common affection. It occurs both as a primary disease (idiopathic atrophy), and as a secondary result of functional interruption or inflammation of the nerve, and of destruction of the retina or of the eye as a whole.

Idiopathic optic atrophy generally takes the form of grey degeneration, the nerve-fibres losing their medulla and accordingly diminishing in bulk, and the nerve being converted into a semi-translucent greyish or greyish-yellow gelatinous cord, containing slender indifferent fibrils in place of its proper substance (LEBER). As the medulla disappears the nerve-fibres are first transformed into pale varicose non-medullated filaments, and these after a time lose their varicosities and become homogeneous. In the initial stage of the disease numerous fat-granule cells appear interspersed between the nerve-fibres, especially at the chiasma and in the optic tracts. Amyloid concretions are also visible here and there, and these likewise are most numerous in the central parts. LEBER was able in one instance to trace their presence as far as the corpora geniculata externa and the surface of the optic thalamus. In more advanced stages both the coarser and the finer fibrous endoneurial septa are more or less thickened, as are also the coats of the vessels; but the fibrous structures generally do not as a rule undergo any great amount of hyperplastic proliferation.

Grey degeneration sometimes makes its first appearance in discrete insular patches scattered longitudinally or over a given transverse section, and confined to particular parts of the nerve, chiasma, or optic tract. As a rule however the atrophic change ultimately becomes diffuse and universal.

The atrophy may be limited to the optic nerve alone; but more frequently it accompanies the like change in the posterior columns of the cord, or is associated with multiple disseminated sclerosis. It has also been observed in cases of progressive paralytic dementia.

Interruption of the functional continuity of the optic nerve from pressure, due for example to tumour, foreign bodies, exudations, or fracture of the optic fissure, or from section of the nerve, is followed by atrophy spreading both ways from the lesion, namely towards the eye and towards the chiasma and optic tract. The descending atrophy extends as far as the retina, destroying its nerve-fibre and ganglionic layers, the other layers usually remaining intact. If the lesion is situated in the optic tract, the atrophic change is long in reaching the eye-ball. Lesions of the corona radiata and of the visual centres in the cortex are also capable of inducing a descending degeneration which, according to MONAKOW, may likewise extend to the optic nerves. Extirpation of the eye, or any destructive lesion of the peripheral terminations of the nerve, in other words destruction of the retina or of the eye-ball as a whole, leads to ascending atrophy, which is most pronounced and most rapid in young animals, as GUDDEN's numerous experiments have shown. But in the case of elderly patients also, ascending atrophy sometimes spreads from the eye to the optic tract within the space of a year (PURTSCHER).

The textural changes associated with degeneration from interruption of functional continuity or from destruction of the peripheral terminations of the optic nerve are, so far as we know, similar to those met with in grey atrophy. Here also the medullary sheaths of the nerve-fibres are the first to disappear. In later stages nothing remains but the fibrous framework of the nerve, without a trace of nerve-substance. *Corpora amylacea* are usually present in varying numbers in the degenerate nerve.

Degeneration of the nerve due to **optic neuritis** appears first at the seat of the inflammation, but (as in cases following the interruption of its functional continuity) the atrophic change is liable to extend throughout the nerve as an ascending or a descending secondary lesion. Thus a neuritis of the trunk of the optic nerve, not originally extending to the papilla, may nevertheless in the end cause atrophy of the latter. Over the segment affected by the initial inflammation, the atrophic nerve usually exhibits not only loss of its nerve-fibres but also perceptible multiplication of its nuclei; in other cases appreciable portions of its structure are replaced by new and abundantly-nucleated connective tissue.

Neuritic atrophy and the consecutive ascending degeneration are sometimes limited to certain tracts within the nerve, such as portions of the superficial bundles or a particular cylindrical sector of the trunk.

New-growths, like inflammation, are also apt to induce atrophy of the optic nerve, from the pressure and the disturbance of the circulation which they cause.

Optic atrophy may lastly be caused by occlusion of the arteries, owing to arteriosclerosis or embolism.

In cases of optic atrophy following circumscribed neuritis, certain morbid appearances are met with that are worthy of notice, inasmuch as they indicate that it is possible for the fibres coming from the yellow spot (*macula lutea*) alone, and in both eyes simultaneously, to undergo degeneration. Apparently the change is due to a local neuritis affecting these fibres, which is followed by degeneration both ascending and descending. The axial portion of the optic nerve, from the optic foramen to the eye-ball, is found to be degenerate, its nerve-fibres having disappeared and its nuclei and fibrous constituents multiplied. The degenerate bundle as it approaches the eye-ball recedes from the axis towards the temporal aspect of the periphery of the nerve-trunk on both sides, occupying an exterior and inferior sector just behind the globe. The atrophic change may also extend, as a simple non-inflammatory degeneration, from the optic foramen on both sides up to the chiasma and the optic tract. UHTHOFF observed this form of neuritic atrophy of the macular bundle in cases of toxic amblyopia due to alcohol, the change being manifested by atrophy of the nerve-fibres, dense interstitial fibrous hyperplasia, multiplication of the nuclei, and the formation of new blood-vessels.

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SCHÜLLER: *Cent. f. prakt. Augenheilk.* V **1881**
TÜRCK: Morbid changes in amaurosis *Z. d. Wiener Aerzte* V **1846**; Pressure on and origin of the optic nerves *ibidem* VIII **1847**
VIRCHOW: Morbid anatomy of the retina and of the optic nerve *V. A.* 10 **1856**

References on Degeneration of the Macular Bundle.

Bunge: *Ueber Gesichtsfeld und Faserverlauf im optischen Leitungsapparat* Halle **1884**
Samelsohn: Retrobulbar neuritis (central amblyopia) *Trans. internat. med. congress* III London **1881**, *A. f. Ophth.* XXVIII **1882**, XXX **1884**
Uhthoff: Toxic amblyopia *A. f. Ophth.* XXXII **1886**, *D. med. Woch.* **1887**
Vossius: *A. f. Ophth.* XXXII **1886**

349. In **myopia** or short-sight the images of near objects alone are sharply defined on the retina, those of remoter objects being blurred. This effect may be due to one or more of three conditions — (1) excessive curvature of the cornea or of the surfaces of the lens, (2) excessive refractive power of the media, (3) abnormal length of the sagittal axis of the eye. As was first pointed out by von Arlt, it is in general the last of these conditions alone that is actually operative. It is especially noteworthy that in myopia the curvature of the cornea is not increased. Keratoconus and cornea globosa may of course also give rise to myopia in certain cases, but ordinary short-sight is unconnected with corneal changes of this kind.

No morbid changes are as a rule apparent in myopic eyes until late in life; though even in youth a high degree of myopia occasionally induces serious alterations in the eye, while in advanced life a certain amount of degeneration is invariably present. The abnormal elongation of the eye-ball is naturally accompanied by stretching and to some extent by thinning of its coats, and these give rise to staphylomatous bulging, chiefly at the posterior pole of the eye in the neighbourhood of the yellow spot and the optic disc. The bulging is generally uniform, the eye becoming egg-shaped with its posterior pole evenly rounded. In some cases, however, the portion between the disc and the yellow spot protrudes as a circumscribed staphyloma. In both forms the axis of the eye-ball may attain a length of thirty millimetres or more, the normal length being from twenty-two to twenty-four millimetres. The causes of the posterior protrusion are not well-understood, but it is probable that it is the sclerotic which gives way: at any rate it is known that in extreme myopia the posterior part of the sclerotic often becomes as thin as paper. The overstretched choroid and pigmentary epithelium likewise become in parts atrophic, and accordingly in highly-myopic eyes the fundus is usually little pigmented. On the temporal side of the optic nerve, moreover, the choroid is very often completely atrophied, the affected portion appearing, when the eye is examined with the ophthalmoscope, as a sickle-shaped patch bordering the temporal side of the disc. As the myopia advances this usually broadens and assumes a crescentic shape (*meniscus* or *conus*): in extreme forms of the affection it constitutes what is called specifically **posterior staphyloma**. When the sclerotic bulges to any considerable extent, the optic disc usually assumes an oblique position relatively to the axis of the eye, so that when observed from the front it looks like an ellipse with its major axis vertical instead of the normal circle (Fig. 551). Large atrophic menisci, when viewed with the ophthalmoscope, have a lustrous bluish-white tint (the colour of the sclerotic), the choroid having entirely disappeared, except perhaps for a few of its vessels or small remnants of its pigment. The meniscus is often bordered by a dark rim due to excessive pigmentation.

The staphyloma may include the half or even the whole of the disc (Fig. 551, annular staphyloma), and sometimes extends towards the yellow spot, where it coalesces with similar atrophic patches of earlier date, that are recognisable with the ophthalmoscope as white spots of rounded or irregular outline. These spots generally represent the final stage of a series of pathological changes in the macular region that are usually comprehended under the term **posterior choroiditis.** It is however the retina that is chiefly involved, and it is not yet certain that the changes in question, which include excessive or irregular pigmentations, haemorrhagic extravasations, and paler atrophic patches of varying size (Fig. 551), are really of inflammatory origin. It is probable that engorgement of the vessels, stretching with relative displacement of the membranes, and the degenerations thereby induced, may likewise play an important part in producing the macular changes characteristic of excessive myopia.

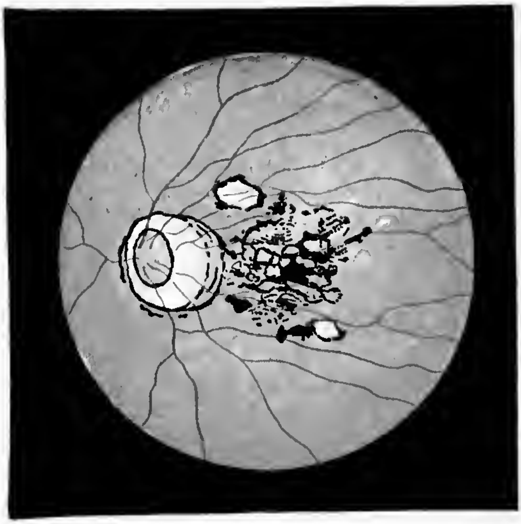
FIG. 551. THE FUNDUS IN EXTREME MYOPIA.

Opacities and softening of the vitreous are especially common in progressive forms of myopia; and these changes are apt to be followed by cataract or detachment of the retina.

Hypermetropia is characterised by abnormal shortness of the sagittal axis of the eye, but it does not lead to any grave changes in the ocular tissues.

With reference to the minute structural changes accompanying myopic posterior staphyloma, it should be noted that, according to KUHNT, the part of the choroid corresponding to the meniscus may become completely atrophic, only the vitreous lamina with a little fibrous tissue remaining and adhering firmly to the sclerotic. Sometimes, however, the atrophy is only partial, the inner layers (the *chorio-capillaris* and the layer of small veins) being alone affected. In the retina overlying the atrophic portion of the choroid, the pigmentary epithelium, the rods and cones, and the outer nuclear layer, are generally absent. KUHNT moreover found, in cases of progressive myopia, isolated patches of actual inflammatory infiltration in the peripheral layer of the choroid. A peculiar condition of the retina in myopic eyes affected with staphyloma was observed ophthalmoscopically by JAEGER and NAGEL, and more recently its minute anatomy has been studied by WEISS and by Duke CARL THEODOR of Bavaria. The traction exerted on the membranes, as the region between the disc and the yellow spot bulges outwards, may drag the retina and choroid over the nasal margin of the papilla, and so give rise to a 'meniscus of supertraction.' The last-mentioned observer found that in the part of the retina thus stretched over the optic papilla the rods and cones were absent, and that the nuclear layers had also undergone certain changes.

As regards the macular affection accompanying myopia, LEHMUS found the substance of the choroid very deeply pigmented and the vessels distended at the part where, during life, a dark patch including the yellow spot had been noticed. In the retina he observed extreme hyperplasia of the pigmentary epithelium, which in the centre of the patch was disposed in several superimposed layers. Between the pigmentary epithelium and the retina lay a stratum of jelly-like exudation, which was thickest at the centre of the patch and contained no cellular elements other than the pigment-cells. WEISS found in a number of myopic eyes that the choroid was thinned about the posterior pole, infiltrated with cells, closely united with the sclerotic, and here and there firmly adherent to the retina also. In cases where the affection of the macular region is more extreme the choroid may be completely atrophied throughout the greater part of its extent.

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Duke CARL THEODOR of Bavaria: *Mittheil. aus der Universitäts-Augenklinik* Munich **1882**
DONDERS: *Anomalies of refraction* (trans. by MOORE) London **1864**
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WEISS: *Nagel's Mittheilungen a. d. ophth. Klinik* part 3 Tübingen **1882**

CHAPTER CXIII

DISORDERS OF CIRCULATION IN THE EYE

350. The vessels of the eye-lids, conjunctiva, sclerotic, etc., which are outside the capsule of the eye-ball, are more subject to rapid fluctuations in the amount of blood they contain than those of the choroid and retina, inasmuch as the latter are exposed to the more or less constant intra-ocular pressure. This pressure serves to maintain the normal form and tension of the capsule, and at the same time exerts a certain amount of compression on the vessels lying within it, which tends to counteract any tendency to undue distension of the choroidal and retinal vessels by arterial congestion or venous engorgement. In the same way it of course tends to impede the intra-ocular circulation, when for example the general arterial pressure falls or when other causes interfere with the afflux of blood to the eye. Under normal conditions two venous channels connected by anastomoses provide for the ready efflux of blood from the eye and orbit; of these the larger passes by way of the sinus cavernosus, the smaller through the anterior facial vein (SESEMANN).

Hyperaemia of the extra-ocular vessels, apart from that associated with inflammation, is in the first place met with in cases where the intra-ocular pressure is morbidly increased (glaucoma), and then concerns the anterior ciliary veins. It depends on the circumstance that the calibre of the *venae vorticosae* that pass obliquely through the sclerotic at the equator of the eye-ball is diminished by the increased intra-ocular pressure, the blood from the choroidal vessels being thereby forced in greater amount through the anterior ciliary veins.

Abnormal distension of the orbital vessels likewise accompanies Graves' disease (KOEBEN, REITH, ROMBERG), and the rare affection known as pulsating exophthalmos, in which the globe protrudes and with its appendages executes pulsatile movements in the direction of the orbital axis. The protrusion and pulsation are due to extreme distension of the orbital veins, and particularly of the superior ophthalmic vein and its branches, and this, according to SATTLER, is in most cases brought about by rupture of the internal carotid artery within the cavernous sinus, permitting the arterial blood to regurgitate into the veins that communicate with the sinus.

Hyperaemia of the intra-ocular vessels is in general apparent only in the iris, the retina, and the optic papilla: the detection of abnormal distension of the choroidal vessels is extremely difficult, and often indeed impossible, either during life or after death. The red tint of the fundus as seen with the ophthalmoscope, which (to say nothing of the visual purple) is referable to the blood-vessels of the choroid and retina, varies in different persons with the depth of the pigmentation of the retinal epithelium and of the choroid. The deeper the pigmentation the more does the red of the fundus tend to pass into a greyish-red or greyish-brown tint. On the other hand, when the pigment is scanty, the chorio-capillaris as well as the larger choroidal vessels are rendered more visible and give the fundus a bright red tint, simulating even in normal conditions of the circulation the appearance of hyperaemia.

Well-marked hyperaemia of the iris (visible externally only in light-coloured eyes) is usually met with either in iritis or as a consequence of new-growths involving the iris.

Non-inflammatory hyperaemia of the vessels of the optic papilla, giving rise to brighter redness mainly of the nasal half of the disc as seen with the ophthalmoscope, is occasionally met with as a result of overstraining the eyes by minute work. Hyperaemia of the disc is also rarely absent in cases of rapidly-progressive myopia. Marked hyperaemia of the papilla or of the retinal vessels is usually however of inflammatory origin.

In cases of venous engorgement the retinal veins are abnormally distended, broadened, and tortuous, the arteries on the contrary being rather narrower than usual. The cause of the engorgement generally lies within the papilla itself: it may be swollen and inflamed, or its central vein may be compressed by glaucomatous pressure, and the return of the venous blood thereby impeded. Engorgement in the territory of the vena cava superior is, for the reason given above, not usually attended by passive hyperaemia of the retinal veins, though according to FÖRSTER and LITTEN these veins are not infrequently found to be greatly distended in patients suffering from emphysema. In certain congenital disorders of the heart and in pulmonary stenosis with general cyanosis, extreme engorgement of the retinal arteries and veins has occasionally been observed (KNAPP, LEBER, LIEBREICH, LITTEN). Great engorgement, accompanied by numerous haemorrhagic extravasations, generally supervenes in thrombosis of the central vein of the retina (MICHEL).

Varicosities of the retinal veins are very rare. They have been seen in glaucoma by LIEBREICH and PAGENSTECHER, and according to the latter are probably due to sclerosis of the vessels combined with the engorgement induced by the glaucomatous pressure.

References on Disorders of Circulation in the Eye.

Förster: *Graefe und Saemisch's Handbuch* vii Leipzig **1877**

Gurwitsch: *A. f. Ophth.* xxix **1883**

Knapp: *Verhandl. des nat.-hist. Vereins* Heidelberg ii **1882**

Koeben: *Inaug. Diss.* Berlin **1855**

Leber: *Graefe und Saemisch's Handbuch* v Leipzig **1878**

Liebreich: *Atlas of ophthalmoscopy* plate ix fig. 3 and plate xi fig. 1 (trans. by Swanzy) London **1885**

Litten: Pulmonary stenosis *Berl. klin. Woch.* **1882**; Emphysema *ibidem* **1881**

Michel: Thrombosis of central vein *A. f. Ophth.* xxiv **1878**

Pagenstecher: *A. f. Ophth.* xvii **1871**

Reith: Case of Graves' disease *Med. Times and Gaz.* ii London **1865**

Romberg and Henoch: *Klin. Wahrnehmungen und Beobachtungen* Berlin **1851**

Sattler: Pulsating exophthalmos *Graefe und Saemisch's Handbuch* vi Leipzig **1880** (with references)

Sesemann: *A. f. Anat. und Physiol.* **1859**

351. **Anaemia** of the eye is of importance chiefly when it affects the optic nerve and the retina, and it is usually in these parts only that it can be definitely made out during life.

General anaemia, unless it is extreme, is not attended by any perceptible diminution of the quantity of blood in the retinal vessels. In extreme cases, however, the optic disc becomes pale, the retinal arteries are greatly contracted, and in general the diameter of the veins is also somewhat diminished, although in some instances the veins appear to be slightly distended. After severe haemorrhage, especially from the intestine or uterus, the optic nerve and the retina of one or (more frequently) of both eyes are liable to become gravely disordered, the morbid change being essentially of the nature of fatty degeneration (Ziegler), and being most apparent at the point where the optic nerve passes through the *lamina cribrosa*. The tissue at this point, and the medullary sheaths of the nerve-fibres in the orbital segment of the nerve, enclose a multitude of large and small oil-globules. In the retina it is mainly the layers of nerve-fibres and of ganglion-cells that are degenerate, the change being most advanced in the papilla, and consisting in fatty degeneration of both the neuroglia-cells and the nerve-elements; the latter are at the same time more or less thickly beset with oil-globules. The process is thus manifestly one of ischaemic degeneration. In the end the nerve-fibres in the retina and optic nerve may entirely disappear and be replaced by connective tissue (Hirschberg).

The anaemia of the retina often observed after toxic doses of quinine and salicylate of sodium is no doubt due to the accompanying fall in the general blood-pressure, whereby the retinal vessels, subjected as they are to the intra-ocular pressure, fail to receive a proper supply of blood (Brunner).

The most extreme form of retinal ischaemia is met with in cases of **embolism** of the central artery of the retina, which is usually a result of valvular cardiac disease, endocarditis, aneurysm of the aorta, or arteriosclerosis.

The embolus generally blocks the trunk of the artery before its subdivision at the papilla, but obstruction may also take place in one of the branches. Very soon afterwards the retinal arteries, as seen with the ophthalmoscope, become almost entirely empty, even the larger branches containing only a slender filament of blood, and the middle-sized and smaller ramifications being as a rule no longer visible. The veins on and about the disc are narrowed, but to a less degree than the arteries; they are generally broader towards the periphery. The disc looks pale and sharply-defined. After a time the arteries and veins become gradually distended, and the retina round the disc and the *fovea centralis* becomes white and opaque. This causes the edges of the papilla to appear blurred, while the whole of the fundus about the yellow spot becomes turbid and milky-looking: within the turbid part lies a cherry-red spot whose centre corresponds to, but is somewhat larger than, the fovea. As the retina over the fovea is normally very thin, the choroid here shines through it, and the red appearance is intensified by contrast with the surrounding turbid opacity. Small haemorrhages into the retina are seen now and then near the papilla. More copious extravasations seem to be prevented by the intra-ocular pressure, which hinders the reflux of blood through the retinal veins.

The retinal turbidity with its red spot may not appear for several days or weeks. Ultimately the opacity disappears and the vessels become slender again, as, with the retina and the optic papilla, they pass into a condition of atrophy. The papilla becomes white, often acquiring a tendinous lustre; its contour however remains sharply-defined.

When a branch of the central artery is occluded by embolism (KNAPP, LANDESBERG) or by syphilitic disease (HAAB), the result may simply be retinal opacity within the anaemic sector, accompanied by hyperaemia and tortuosity of the veins, or retinal haemorrhage may take place.

Thrombosis of the central artery and its branches produces alterations similar to those resulting from embolism. It is probably in most cases due to disease of the vessel-wall (such as syphilitic arteritis).

In atrophy of the optic nerve and retina great shrinkage of the retinal vessels usually takes place; it may become so extreme that the filament of blood they contain is no longer visible. The change depends on progressive obliteration of the vessels with thickening of their walls.

References on Anaemia of the Retina.

BRUNNER: Amaurosis from quinine, *Inaug. Diss.* Zürich **1882** (with references on retinal ischaemia from quinine and salicylic acid)

FÖRSTER: *Graefe and Saemisch's Handbuch* VII Leipzig **1877**
GRAEFE, ALF.: *A. f. Ophth.* VIII **1862**
HEDDAEUS: *Klin. Monatsbl.* IV **1866**
HIRSCHBERG: Anaemia after loss of blood *Z. f. klin. Med.* IV **1881**
ROTHMUND: *Klin. Monatsbl.* IV **1866**
ZIEGLER: Amaurosis after loss of blood *Ziegler's Beiträge* II **1887** (with references)

References on Thrombosis and Embolism of the Retinal Vessels.

FISCHER: *Embolie der Art. central. Retinae* Leipzig **1891** (with references)
HAAB: Syphilitic arteritis *Helmholtz's Festschrift* Stuttgart **1891**
KNAPP: *A. f. Augen- u. Ohrenheilk.* and *A. of Ophth. and Otol.* I **1869**
LEBER: *Graefe and Saemisch's Handbuch* V Leipzig **1877**
MANZ: Embolism *Helmholtz's Festschrift* Stuttgart **1891**
MICHEL: Venous thrombosis *A. f. Ophth.* XXIV **1878**

352. **Haemorrhage** in and about the eye may result in the first place from wounds of the most various kinds; thus even a smart blow or knock on the eye-ball sometimes causes effusion of blood into the aqueous or vitreous humour. More frequently the choroid is lacerated, particularly in the neighbourhood of the optic nerve, the parts of the choroid and retina adjacent to the rent becoming thereby infiltrated with blood.

Spontaneous haemorrhage takes place generally into the conjunctiva and retina, rarely into the choroid.

Haemorrhage into the conjunctiva may attend excessive engorgement of the branches of the superior vena cava, such as is caused by violent coughing, vomiting, epileptic convulsions, or the lifting of heavy loads. In such cases the blood usually escapes under the ocular conjunctiva (*hyphaema conjunctivae*) in the form of red spots of various sizes. In elderly people who suffer from vascular atheroma, cardiac lesions, or cardiac hypertrophy, conjunctival haemorrhage sometimes makes its appearance without any assignable cause, and is then often a precursor of cerebral haemorrhage.

Haemorrhage into the choroid is rare: it is generally single, but is sometimes of considerable magnitude. As a rule it occurs in aged or anaemic persons.

Haemorrhage into the retina is liable to accompany extreme engorgement (REICH) and inflammations of the membrane; but it is oftener due to sclerotic, atheromatous, fatty, amyloid (ALT), or hyaline (OELLER) degeneration of the vessels: in other cases it is associated with diabetes or nephritis, and leukaemic or simple anaemia. It gives rise to a spotted condition of the retina, which is usually described as haemorrhagic retinitis.

In cases of atheromatous degeneration the haemorrhages are usually confined to one eye, but they are multiple, and the retina may be studded over with innumerable small and occasionally coalescent spots varying in colour from pale-pink to dark-red or black. In the layer of nerve-fibres the blood is apt to spread in a radial manner, forming [illegible] lines or streaks; in the deeper layers the extravasations are more rounded in outline. Occasionally the blood intrudes between the retina and the vitreous, or penetrates into the latter.

In thrombosis of the central vein, in addition to great distension and tortuosity of the retinal veins, swelling and hyperaemia of the papilla, and haziness of the optic disc, copious haemorrhage is not uncommon.

In **diabetes mellitus** both scattered haemorrhages and small white patches of degeneration resembling those of albuminuric retinitis are met with. Opacities of the vitreous are frequently seen, and these also are no doubt dependent on retinal haemorrhage.

In **nephritis**, besides the other changes characteristic of albuminuric retinitis (Art. 369), retinal haemorrhages in the form of streaks or of rounded spots also make their appearance.

In **leukaemia** retinal haemorrhages are observed in about a third of the cases (LEBER), and usually in both eyes. White spots also appear in the fundus, due partly to varicose enlargement of the nerve-fibres (VON RECKLINGHAUSEN), partly to leukaemic infiltration, and partly to fatty degeneration. Especially characteristic is the appearance of yellowish-white spots surrounded by a haemorrhagic areola and projecting slightly above the surface of the retina; they are situated for the most part in its anterior portions, sometimes about the yellow spot also, and are interspersed with small round haemorrhagic spots. The pale patches consist of masses of red and white blood-corpuscles. The larger ones occupy the entire thickness of the retina, the smaller ones lie in the inner layers. DEUTSCHMANN, in addition to these extravasations, observed some enlargement of the radial nerve-fibres and sclerotic thickening of the others.

The vessels, as seen with the ophthalmoscope, are abnormally pale, the veins being pink and the arteries pale-orange, while the fundus often assumes a pale yellowish-red tint. The veins are usually broad, and at times appear bordered by white marginal streaks, due to the accumulation of leucocytes along their course. MICHEL found that in one case thrombosis of the central vein was present, and in another thrombosis of the superior ophthalmic vein.

In grave **pernicious anaemia** retinal haemorrhages are almost always present; the retina may indeed be the only structure into which haemorrhage takes place. The patches are pale-red in colour and linear or rounded in form: in size they are sometimes merely punctiform, in other cases they are of considerable extent. The papilla is pale, and the veins tortuous and widely distended. Here and there isolated white spots are seen, and the larger extravasations are occasionally white in the centre. The haemorrhagic patches occupy the inner layers of the retina, most frequently the layer of nerve-fibres and the internal granular layer (QUINCKE). The whitish centre consists of finely granulated matter or of lymphoid cells. UHTHOFF observed in several cases globular and lustrous fusiform varicosities on the nerve-fibres, and highly-refractive colloid and finely-granular masses lying in the internal granular layer.

Retinal haemorrhages are also met with in purpura, phosphorus-poisoning (NIEDERHAUSER, LITTEN), intermittent fever (VON KRIES), and diseases of the liver associated with jaundice, and also in connexion with extensive burns of the skin (KNIES, WAGENMANN).

Both in the retina and in the conjunctiva the extravasated blood is after a time re-absorbed, usually leaving no perceptible alteration behind: abundant haemorrhagic infiltration of the retina may however lead to atrophic changes in the membrane.

References on Vascular Degeneration in Intra-ocular Haemorrhage.

ALT: Amyloid degeneration *The human eye* New York **1880** and *Histologie des Auges* Wiesbaden **1880**
LEBER: *Graefe und Saemisch's Handbuch* v Leipzig **1877**
MANZ: *Verhandl. d. Naturf.-Gesellsch. zu Freiburg i. B.* IV
MICHEL: Thrombosis of the central retinal vein *A. f. Ophth.* XXIV **1878**
OELLER: Hyaline degeneration *V. A.* 86 **1882**
PAGENSTECHER: *A. f. Ophth.* XVII **1871**
REICH: *Cent. f. Augenheilk.* **1883**

References on Retinal Changes in Leukaemia.

DEUTSCHMANN: *Klin. Monatsbl.* XVI **1878**
LEBER: *Klin. Monatsbl.* VII **1869**
MICHEL: *D. A. f. klin. Med.* XXII **1878**
OELLER: *A. f. Ophth.* XXIV **1878**

References on Retinal Haemorrhage in Anaemia.

BIERMER: *Correspbl. f. Schweizer Aerzte* **1872**
HORNER: *Klin. Monatsbl.* XII **1874**
VON KRIES: *A. f. Ophth.* XXIV **1878**
LITTEN: Relation to diseases of the liver *Z. f. klin. Med.* V, and *D. med. Woch.* VIII **1882**; *Berl. klin. Woch.* **1879**
MÜLLER, H.: Progressive pernicious anaemia etc. *Inaug. Diss.* Zürich **1877**
NIEDERHAUSER: Aetiology and significance of retinal apoplexy *Inaug. Diss.* Zürich **1882**
QUINCKE: Pernicious anaemia *Volkmann's klin. Vorträge* no. 100 Leipzig **1876**
UHTHOFF: *Klin. Monatsbl.* XVIII **1880**
WEIGERT: *V. A.* 79 **1880**
ZIMMERMANN: *D. A. f. klin. Med.* XIII **1874**

References on Retinal Haemorrhage following Burns.

KNIES: *Grundriss d. Augenheilkunde* Wiesbaden **1892**
WAGENMANN: *A. f. Ophth.* XXXIV **1888**

353. **Oedema** of the lids is observed most frequently in the dropsy of Bright's disease and in trichinosis. In the conjunctiva it is most likely to occur in the ocular portion of the membrane,

constituting so-called **chemosis**, as a result of purulent inflammation of the lids (especially in *hordeolum* or 'stye'), or of the choroid and ciliary body: it also appears in connexion with suppurative irido-choroiditis, in incipient inflammation of the entire globe (panophthalmitis), and in ophthalmia or conjunctivitis (particularly gonorrhoeal ophthalmia). Extreme chemosis has also been observed in cases of pyaemic thrombosis of the cavernous sinus and of the orbital veins.

The intra-ocular tissues rarely exhibit signs of simple oedema, although inflammation and engorgement at the fundus are often accompanied by oedematous swelling of the papilla and surrounding parts of the retina. Cystic degeneration of the retina (Art. 347) is also sometimes described as oedema (IWANOFF). The detachment of the bacillary layer observed in connexion with slight detachment of the retina is regarded by LEBER, who first called attention to it, as due to retinal oedema. In these cases the bacillary layer is over a considerable area separated from the external limiting membrane by a thin stratum of liquid.

References on Oedema of the Conjunctiva.

BURNETT: *A. f. Augenheilk.* x and *A. of Ophth.* x **1881**
SCHIESS: *Klin. Monatsbl.* x **1872**
ZEHENDER: *Klin. Monatsbl.* VIII **1870**

CHAPTER CXIV

INFLAMMATIONS OF THE EYE

354. **Blepharitis** or inflammation of the eye-lids is commonest at their ciliary margins (*blepharitis ciliaris*). The margins containing the eye-lashes and the various glands embedded in the palpebral tissue are the parts chiefly concerned. Various forms of the affection are distinguished, according to the appearances they present, as seborrhoea, eczema, and acne.

Seborrhoea (*blepharitis squamosa*) is generally a local manifestation of a disease affecting the entire hairy scalp and known as *seborrhoea sicca* or *pityriasis furfuracea capillitii* (Art. 171). As in the cutaneous affection, its characteristic symptom is the formation of sebaceous scales, which overlie the portion of the lid whence the eye-lashes spring. At the same time the margins of the lids become swollen and thickened, and the circumglandular tissue infiltrated. In the course of the disease the lashes drop out, and those that follow them become successively finer, shorter, and less pigmented. In the end some of them are not replaced at all, so that at times the reddened edges of the lids are bordered only by a sparse row of lanuginous filaments. The outer horny layer of the epidermis is often exfoliated underneath the sebaceous scales. In young patients the disease at first presents the appearance of *seborrhoea oleosa*, thick yellowish sebaceous crusts being formed between the lashes. In the later stages these are generally succeeded by the characteristic furfuraceous scales.

Eczema of the lids (*blepharitis ulcerosa*) is characterised by the formation of pustules, which at the points where they occur destroy the epidermis, and sometimes also the papillary layer of the corium, so giving rise to small round ulcers between the lashes. The ulcers are commonly covered over with crusts and scabs, and are not apparent until the latter are removed. When the ulcers are deep and wide the eye-lashes at the affected points are irreparably destroyed, so that in the end gaps in the line of the cilia remain as evidence of the antecedent disease.

Acne of the lids presents exactly the same appearances as acne occurring in the skin of other parts, the affection having its seat in and about the sebaceous glands and the follicles of the eye-lashes. The inflammatory nodule is usually described as a *hordeolum* or stye. It may be artificially produced by introducing the *Staphylococcus pyogenes aureus* into the conjunctival sac (HAAB). When the inflammation starts in the meibomian glands, the nodular infiltration is situated in the tarsus (*hordeolum meibomianum*), and attains a greater size than the ordinary stye. The affection accordingly gives rise to a circumscribed thickening of the tarsus, and this in some cases undergoes suppuration.

When the excretory duct of a meibomian gland is occluded, retention of the secretion and chronic inflammation ensue, giving rise to the peculiar nodular growth known as **chalazion.** This consists of soft greyish-red granulation-tissue, here and there beset with giant-cells, and containing in the centre a certain amount of serous or muco-purulent liquid. Chalazion is generally subsequent to catarrhal conjunctivitis (HAAB). It is probable that the conjunctivitis leads in some unknown way to the infection of the meibomian glands.

Inflammation of the **tarsus** is not often met with; it is most apt to make its appearance in connexion with scrofula and syphilis.

The **lacrimal gland** is rarely the seat of either chronic or acute inflammation. The latter may however issue in suppuration. But acute inflammation of the lacrimal sac and the connective tissue about it (**dacryocystitis**) is common, and is usually due to obstruction of the nasal duct (dacryostenosis).

355. The forms of **conjunctivitis,** or inflammation of the conjunctiva, may be divided into two groups, namely such as usually spread diffusely over the entire mucous membrane, and such as tend rather to occur in circumscribed patches, single or multiple. To the latter group belong eczema and the syphilitic and tuberculous inflammations; to the former the several varieties of catarrh, and croupous, diphtheritic, gonorrhoeal, and trachomatous conjunctivitis or ophthalmia.

Simple **catarrh** of the ocular conjunctiva (*conjunctivitis catarrhalis*), like catarrh of other mucous membranes, leads to swelling and hyperaemia of the mucosa, accompanied in the first stage of the affection by a muco-serous, and afterwards by a muco-purulent secretion. In the more advanced stages of the catarrh the palpebral conjunctiva, especially at the fornix where the mucous membrane of the lid passes on to the eye-ball, usually becomes longitudinally corrugated and roughened (villous swelling). The mucosa between the minute furrows intersecting in all directions that are normally present in the membrane becomes thickened by multiplication of its lymphoid cells and by vascular hyperaemia, and in this way papilla-like prominences are formed and the intervening epithelial furrows are deepened. Purulent catarrh may make its appearance even in new-born infants, and is then apt to be mistaken for blennorrhoea or gonorrhoeal ophthalmia.

Croupous conjunctivitis is rare; it usually attacks the palpebral rather than the ocular membrane. The croupous false-membrane is readily separable from the mucosa, which is hyperaemic and denuded of its superficial epithelium, but the deeper layers are not eroded. The affection appears to be of diphtherial origin. In several cases UHTHOFF detected the diphtheria-bacillus in the croupous membrane.

In **diphtheritic conjunctivitis** the membrane is converted into a dry, tough, greyish mass, not only the surface epithelium but the mucosa and submucosa undergoing necrosis. By suppuration and sloughing of the infiltrated structures, ulcerous erosion and loss of substance take place, and in case of recovery can be made good only by granulation and cicatrisation.

Diphtheritic inflammation of the conjunctiva is very apt to induce ulceration of the cornea at its margin and centre. The ulcers assume a peculiar yellowish or yellowish-brown tint, as if the corneal tissue had there been directly invaded by the microbes.

Gonorrhoeal conjunctivitis of infants (*blennorrhoea neonatorum*) and of older children and adults arises from infection of the conjunctiva with genital or ocular secretions that contain *Gonococcus*. It is an intense inflammation resembling very severe purulent catarrh, and often leads to secondary destruction of the skin of the lids. The conjunctiva does not as a rule undergo permanent cicatricial transformation, though its appearance may be greatly altered by thickening, corrugation about the fornix, villous swelling of the palpebral portion, and infiltration and oedema of the sclerotic portion. Erosion of the cornea often takes place from the action of the profuse purulent secretion. In new-born infants it is usually the centre of the cornea that is eroded; in adults the periphery is more apt to be attacked, the suppurating ulcer thus produced being very liable to break through into the anterior chamber and so to destroy the entire cornea.

The penetration of the gonococci into the superficial layers of the mucosa is followed by dense cellular infiltration, and the emigrant cells that yield the secretion detach and extrude the greater part of the epithelium. This is afterwards reproduced, in the first instance in the form of pavement-epithelium (BUMM).

References on Croupous and Diphtheritic Conjunctivitis.

VON ARLT: *Clinical studies on diseases of the eye* (trans. by WARE) Edinburgh and Philadelphia **1885**
HORNER: *Gerhardt's Handbuch der Kinderkrankheiten* v part 2 Tübingen **1882**
MANZ: Croupous ophthalmia *A. f. Augenheilk.* XIV and *A. of Ophth.* XIV **1885**
UHTHOFF: *Berl. klin. Woch.* **1893** and **1894** (with references)

References on Blennorrhoea Neonatorum and Gonorrhoeal Ophthalmia.

BUMM: *Der Mikroorganismus der gonorrhoischen Schleimhauterkrankungen* (*Gonococcus-Neisser*) (1st and 2nd edition) Wiesbaden **1885–7**
HAAB: *Horner's Festschrift* **1881**, *Correspbl. f. Schweizer Aerzte* **1885**
KRAUSE: *Cent. f. prakt. Augenheilk.* **1882**
NEISSER: *Cent. f. med. Wiss.* **1879**, *D. med. Woch.* **1882**, *Breslau. ärztl. Zeit.* **1886**
SATTLER, LEBER, HIRSCHBERG: *Klin. Monatsbl.* (Supplement) XIX **1881**

356. **Chronic blennorrhoea** or **trachoma**, also known as Egyptian ophthalmia or *conjunctivitis granulosa*, is a peculiar infective inflammation affecting the conjunctiva, but not met with in other mucous membranes. Notwithstanding its wide prevalence in many countries, the textural changes that take place in the conjunctiva in this disease are still imperfectly known and variously interpreted. Probably no satisfactory account of the process can be looked for until the origin and mode of conveyance of the virus are determined, and this has not yet been accomplished.

The affection is chronic, with acute exacerbations, and the pathological changes it induces are at first confined to the palpebral conjunctiva, spreading thence to the tarsus and to the ocular conjunctiva and cornea. In the palpebral conjunctiva the process gives rise to diffuse cellular infiltration. The mucous membrane is thereby thickened to six or eight times its normal depth, but the thickening is not uniform. The normal epithelial depressions, in the form of minute reticulate furrows, grooves, and tube-like pits, persist and increase in depth in proportion as the adenoid layer of the mucosa between them is thickened and so elevated. In this way are formed a number of papillary elevations (Fig. 552 *f*) intersected by deep (*a b d*) and often bifurcating (*c*) clefts lined with epithelium, the bottom of each occupying the place of one of the original furrows of the normal mucosa.

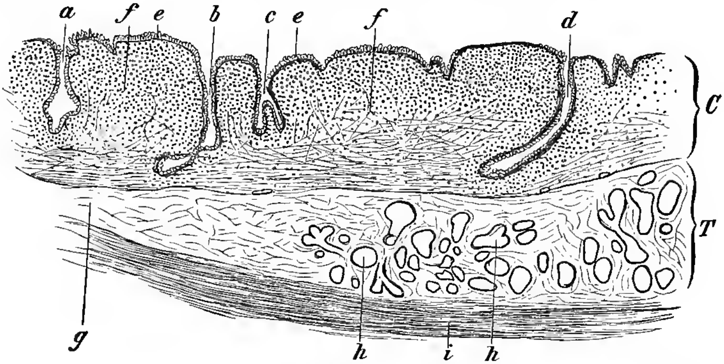

FIG. 552. TRACHOMA OF THE CONJUNCTIVA.

(*Preparation by* IWANOFF *from the collection of Prof.* HORNER: *slightly magnified*)

T	tarsus
C	conjunctiva palpebrae superioris
a b c d	invaginations of the epithelium (from deepening of the normal grooves and depressions)
e	epithelium of the conjunctiva
f	proliferous conjunctival tissue
g	upper border of the tarsus *T*
h	acini of meibomian glands
i	layer of muscle and skin on the front of the tarsus

Another structural element characteristic of the affection is the trachomatous granule, which forms a circumscribed adenoid growth resembling a lymph-follicle; the granules appear on the surface as sago-like greyish-red nodules varying from 1 to 4 millimetres in diameter. These project somewhat above the level of the mucous membrane, and are most numerous and prominent in the fornix; in the tarsal conjunctiva they are more deeply embedded in the tissue and are usually smaller. According to JACOBSON the granules are circumscribed inflammatory growths of the same structure as the diffuse inflammatory hyperplasia of the rest of the affected mucosa. According to LEBER, the granules constitute the specific morbid structure characteristic of trachoma, being the product of a special infective process like the granulomatous nodules of tuberculosis, syphilis, and lupus.

When trachoma persists for a time, the conjunctiva usually assumes a cicatricial appearance that is met with in no other disease of the conjunctiva (with the exception perhaps of certain forms of diphtheritic ophthalmia). The tarsal cartilages at the same time undergo fatty degeneration and atrophy, and their form (particularly in the case of the upper lid) is altered by the shrinking of the mucous membrane, the edge of the lid turning inwards and the tarsus curving into a trough-like shape. The lashes are thus forced against the eye-ball (trichiasis), and sometimes the edge of the lid becomes so incurved that the skin is drawn round it on to the inner surface (entropion), the effect resembling that sometimes produced by extensive burns and corrosions, or by severe diphtheritic ophthalmia.

When the disease spreads to the ocular conjunctiva and the cornea, these also become the seat of superficial cellular infiltrations, trachomatous granules, and papillary excrescences. The subepithelial cellular proliferation and infiltration advance in the form of a vascular pannus from the upper edge towards the centre of the cornea, and in the end may overspread it entirely. The vascular film covering the cornea may be from 1 to 2 millimetres thick, and is probably an extension of the trachomatous overgrowth of the conjunctiva: it is therefore (VON ARLT and others) to be regarded as a specific inflammatory product and not as a mere effect of persistent friction.

Concerning the amyloid degeneration of the conjunctiva associated with trachoma, see Art. 342.

Follicular catarrh of the conjunctiva is from the anatomical point of view closely allied with trachoma, for although the affection is not contagious, or but slightly so, it results in the formation of granulations that are very much like those of trachoma; indeed in the early stages it is impossible to distinguish between them. The deeper textural changes characteristic of trachoma are, however, rarely if ever met with in follicular catarrh, the ultimate cicatrisation and contraction never supervening. The granulations in follicular catarrh appear chiefly in the lower fornix, tend to disappear spontaneously without ulceration, and leave no trace behind. Follicular catarrh may also be produced by certain drugs, such as atropine, and is often the outcome of chronic irritation of the mucosa by some noxious agency like the virus of tuberculosis.

References on Trachoma.

JACOBSON: *A. f. Ophth.* XXV **1879**
KUHNT: *A. f. Augenheilk.* X and *A. of Ophth.* X **1881**
LEBER: *A. f. Ophth.* XXV **1879**
MICHEL: Specific micrococci *A. f. Augenheilk.* XVI **1886**
RÄHLMANN: *A. f. Augenheilk.* XXI **1881–2**
SAEMISCH: *Graefe and Saemisch's Handbuch* IV **1876** (with references)

357. Another peculiar non-infective inflammatory affection of the conjunctiva is known as **spring catarrh** (*phlyctaena pallida*) or gelatinous infiltration of the limbus (VON GRAEFE). This disease usually attacks both eyes, reaching a climax in the warmer months and often disappearing entirely in the winter. It generally recurs for several years successively, but finally ceases spontaneously. The pathological changes it induces are of three kinds. On the nasal and temporal sides of the corneal margin, and particularly over the portion of the eye-ball exposed when the lids are open, appears a greyish-red turbidity with swelling of the limbus, resembling somewhat an eczematous eruption. The swelling is not, however, pustular in form, but has rather the appearance of a flat rampart-like elevation, with a smooth or at most slightly uneven non-ulcerous surface. The injection of the adjacent conjunctiva is moreover less intense than would accompany similar pustules of eczema affecting the corneal margin. The palpebral conjunctiva, especially that of the lower lid, nearly always exhibits a greyish superficial turbidity, as if it were overlaid with a thin film of milk (HORNER). When the disease has persisted for some time, the tarsal conjunctiva of the upper lid, and it may be of the lower lid also, appears studded with large flattened and rounded or pedunculated granulations, isolated or crowded together, and from 1 to 3 millimetres broad, which are of the same tint as the milky portions of the conjunctiva: VON GRAEFE described these as pavement-granulations.

In chronic cases, the cornea on its nasal and temporal sides acquires a narrow line of opacity, parallel to which a second opacity like a short arcus senilis often makes its appearance.

Over the marginal swelling of the limbus, the superficial opacities of the palpebral conjunctiva, and the pavement-granulations, the epithelium is hyperplastic, and not only covers the growths to an unusual depth, but also sends long conical processes deep into the underlying tissue. This overgrowth is usually most exuberant on the larger swellings about the limbus, and on the growths studding the conjunctiva of the upper lid, assuming there something of the appearance of an epitheliomatous proliferation. The adenoid tissue of the mucosa is thickly infiltrated with round-cells, and in it new connective tissue is often abundantly produced. No follicular structures like the granules of trachoma are ever developed. It has not yet been proved that the affection is of microparasitic origin.

References on Spring Catarrh.

BURNETT: *A. f. Augenheilk.* XI **1882** and *A. of Ophth.* X **1881**
KNUS: *Inaug. Diss.* Zürich **1889**
REYMOND: *Annali di ottalm.* IV **1874**
SAEMISCH: *Graefe and Saemisch's Handbuch* IV **1876**
SCHIELE: *A. f. Augenheilk.* XIX **1889**
UHTHOFF: *Klin. Monatsbl.* (supplement) XX **1882**
VETSCH: *Inaug. Diss.* Zürich **1879**

358. The commonest of the circumscribed inflammations of the conjunctiva, and indeed the commonest of all the inflammatory affections of this membrane, is **phlyctenular conjunctivitis** (lymphatic or scrofulous ophthalmia), an affection which might perhaps be more correctly described as **conjunctival eczema** (HORNER). It is characterised by the appearance on the ocular conjunctiva, particularly about the limbus or zone immediately surrounding the cornea, of papules or pustules (*phlyctaenulae*) either isolated or in considerable number, and varying from the size of a grain of sand to that of a pin's head (though they occasionally measure as much as 5 millimetres in diameter), around which the conjunctiva is intensely injected. The summit of each papule soon turns whitish, disintegration invariably setting in at this point, and giving rise to a small shallow ulcer, with a greyish-white floor. The inflammation that accompanies the eruption of pustules is usually confined to their immediate neighbourhood. The efflorescence of a large number of minute *phlyctaenulae* is accompanied by muco-purulent catarrh with injection and swelling (eczematous catarrh of HORNER). Pustules are rarely if ever formed in the conjunctiva of the lids and fornix. The cornea, however, may be the seat of a precisely similar circumscribed inflammation (Art. 360).

The conjunctival eruption is described by HORNER as consisting of rounded greyish-red elevations of very variable size, microscopic sections of recent cases showing that over each prominence the epithelium remains intact, while the subepithelial tissue is packed tight with a solid aggregation of round-cells. It would therefore appear that the term *phlyctaenula* (or vesicle) is inappropriate, the expressions papule and pustule being preferable from a histological point of view.

As the eczema runs sometimes an acute and sometimes a chronic and recurrent course, the eruption is singularly variable in appearance, especially as at one time the conjunctiva, at another the cornea, and again both together, are liable to become involved, and all this at irregular intervals and with frequent recurrences. Eczematous eruptions upon the face, nose, ears, etc. often make their appearance at the same time.

Variolous pustules of the conjunctiva generally appear at the lower edge of the cornea about the limbus, and as in the case of eczema these sometimes form the starting-points for destructive lesions of the cornea. Such lesions at times assume the form of marginal ulceration or of deep-seated purulent infiltration, and lead to perforation or staphyloma, to suppurative choroiditis, and even to panophthalmitis (Horner).

Pemphigus of the conjunctiva is rare, but in certain cases it terminates in complete destruction of the mucous membrane (xerophthalmia, Art. 342).

Circumscribed lesions of the conjunctiva due to **syphilis** (primary and secondary ulcers and gummata), **tuberculosis** (lupus), or **leprosy** are very rare. Tuberculosis produces on the conjunctiva of the lids and eye-ball somewhat broad and flattened excrescences, not unlike the fungous granulomata of synovial membranes, with a red uneven or granular surface. When the lesions are extensive the granulations are sometimes pitted with ragged ulcers of various sizes, on the floors of which grey or caseous tubercles are visible. By the coalescence of a number of such tubercles large agglomerate nodes are sometimes formed. In the fornix follicular granules are not infrequently produced.

References on Circumscribed Conjunctivitis.

Albrand: Pemphigus *Klin. Monatsbl.* **1894** (with full references)
Amiet: Tuberculosis *Inaug. Diss.* Zürich **1887** (with references)
Bäumler, E. and Gelpke: Pemphigus *Klin. Monatsbl.* xxiii **1885**
Baumgarten: Tuberculosis *A. f. Ophth.* xxiv **1878**
Haab: Tuberculosis *A. f. Ophth.* xxv **1879**
Horner: Eczema and variola *Gerhardt's Handb. d. Kinderkrankh.* v Tübingen **1882**
Köster: Tuberculosis *Cent. f. med. Wiss.* **1873**
Manz: Tuberculosis *Klin. Monatsbl.* xix **1881**
Pagenstecher and Pfeiffer: Lupus *Berl. klin. Woch.* **1884**
Rhein, Wagenmann: Tuberculosis *A. f. Ophth.* xxxiv **1888**
Stölting: Tuberculosis *A. f. Ophth.* xxxii **1886**
Schweigger: Pemphigus *A. f. Augenheilk.* and *A. of Ophth.* xiii **1884**

359. **Inflammation of the cornea.** The cornea, though it is non-vascular, is very frequently the seat of inflammation, which is manifested by grey or by yellowish turbidity of the affected parts and by circumcorneal injection, in other words by hyperaemia of the adjacent conjunctival and subconjunctival vessels. The inflammation may be diffuse or disseminated; it may involve at one time the superficial, at another the deeper layers of the cornea, or it may gradually extend inwards from the surface. In the course of the affection new blood-vessels are often formed within the corneal tissue.

Every inflammation of the cornea is attended by the immigration of white blood-corpuscles into the corneal tissue. These either reach the lymphatic spaces of the cornea directly from the adjoining parts of the sclerotic and conjunctiva, or penetrate from the conjunctiva through some erosion of its anterior surface (COHNHEIM). In the former case the immigration takes place through the margin of the cornea or some portion of it; in the latter through some accidental spot on its surface. The former is no doubt the commoner mode of entrance, but in all corneal inflammations arising from catarrhal or purulent affections of the conjunctiva it is probable that a certain amount of immigration from the conjunctival sac takes place.

Although the texture of the cornea is very compact, the ingress of leucocytes is facilitated by the numerous ramifications of the interlamellar canals which permeate it, and which even under normal conditions contain a few amoeboid cells, especially in the marginal region. The chief source of the immigrant leucocytes is the zone of capillary loops in the limbus between conjunctiva and cornea, a strip of conjunctival tissue 1 to 1·5 millimetres in width which overlaps the corneal edge. It contains a multitude of fine capillaries running towards the cornea, which after dividing and subdividing dichotomously all turn back again, and in this way form a close network of terminal vascular loops. From these and the subconjunctival vessels the leucocytes make their way into the corneal canals, and as in these they often lie closely packed, the trains of immigrant cells so formed may occasionally be detected in the living subject under suitable (focal) illumination as short bright streaks, which lie at different levels and as they intersect at right angles (following the general course of the canals) produce a delicate lattice-like pattern. Microscopical examination (Fig. 553 *E*) reveals the presence in the corneal spaces of leucocytes that appear round or spindle-shaped, according to the breadth of the channels in which they lie. Here and there some of them are observed to have become disintegrated (*K*).

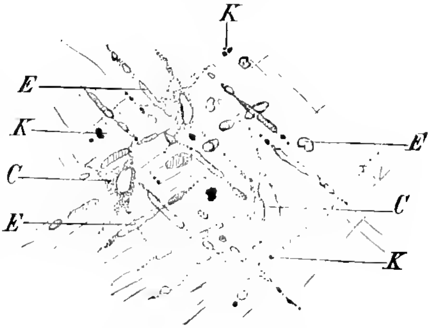

FIG. 553. KERATITIS.
(*Haematoxylin-staining* : × 300)

C corneal cells
E nuclei of pus-corpuscles of different forms
K disintegrated nuclei of pus-corpuscles

In the early stages of the inflammation the fixed corneal cells (*C*) retain their normal appearance: as a rule it is only at the site of the initial lesion, whether wound or point of infection, that they speedily undergo disintegration. When the accumulation of pus-corpuscles goes on for a long time the fibrils and fasciculi of the connective tissue are damaged, eroded, and loosened; but when the infiltration is transient the corneal ground-substance usually undergoes no pathological change.

In every form of keratitis the surface of the cornea over the infiltrated portions loses its brilliant lustre and becomes dull and uneven, as if it had been breathed upon. The turbidity is due chiefly to degeneration and loosening of the overlying epithelial cells. In the preparation of specimens these are found at the dull places to fall off more readily than the normal epithelium. Where the epithelial layer persists, its anterior surface is no longer smooth, but somewhat wavy and uneven, and the mutual cohesion of the cells is impaired.

So long as the pus-corpuscles infiltrating the cornea are not too closely crowded together the turbidity they produce is of a greyish tint; but when they are thickly aggregated they give rise to a yellow discoloration. Circumscribed purulent infiltrations of the ground-substance are somewhat inappropriately described as **abscesses**, though no pus-containing cavity is produced. When the greyish nebulosity or the yellow discoloration spreads over the entire cornea, the inflammation is termed interstitial, parenchymatous, or **diffuse keratitis.**

Any one of the circumscribed superficial inflammations, whether purulent or not, may lead to an erosion or **corneal ulcer** (*ulcus corneae*). This is true not only of traumatic keratitis, but also of other disseminated inflammatory lesions, such as eczema and herpes. Deep ulcers may penetrate into the anterior chamber, especially when they are of a suppurative character.

Purulent ulceration and infiltration of the cornea are usually due to microbic infection. The toxins produced by the micro-organisms not only induce necrosis of the subjacent tissue (Fig. 557) and copious immigration of leucocytes, but as they diffuse into the anterior chamber set up fibrinous and purulent inflammation of the iris and ciliary body, and thus give rise to **hypopyon,** a more or less abundant accumulation of pus at the lower part of the chamber. The components of this pus are therefore not derived from the cornea, the corneal pus-corpuscles being unable to migrate through the membrane of Descemet (Leber). The membrane is also impermeable by the pyogenic micrococci, and thus an ordinary hypopyon is generally free from germs; but it sometimes gives way under the stress of copious purulent infiltration of the cornea, and then corneal pus mingles directly with the exudation in the anterior chamber (Silvestri).

The process of repair and the secondary changes following upon corneal inflammation consist in the formation of new vessels, the replacement of the tissue lost by ulceration, and the production of cicatrices, the latter giving rise to permanent opacities of the cornea. By regenerative multiplication of the surviving corneal cells those that have perished are replaced by new ones, and in this way an ulcerous erosion may be gradually filled up again, and the normal even curvature of the corneal surface be restored (Fig. 554). The epithelium is in general reproduced more rapidly than the ground-substance, so that the excavation is first covered in with epithelium (Fig. 554 *E*) and afterwards filled up by the proliferation of the proper corneal tissue beneath. The newly-formed fibrils resemble those of the normal cornea, but they are not quite normal in their disposition and transparency; the resulting scar-like spots are therefore somewhat turbid or opaque, and are known as *maculae corneae.*

In man the repair of an ulcerous erosion of any considerable size is attended by the development of new blood-vessels, one or more of which appears to run toward the ulcer from the edge of the cornea. They spring from the marginal capillary loops (ARNOLD), run usually close to the surface of the cornea, and subdivide dichotomously at the ulcer. Vascularisation of this kind appears to be indispensable for the efficient production of the reparative material, the rapidity with which the new tissue is elaborated depending upon the rate at which the new vessels are formed.

When fresh foci of inflammation are successively produced, and give rise to a large number of infiltrated spots and superficial ulcers of various sizes, the cornea or some considerable portion of it is liable to become pervaded by radial dichotomous vessels, nearly all of which run in its anterior layers. This condition is known as **pannus**. More deep-seated infiltrations (in diffuse keratitis) lead to vascularisation of the deeper layers also.

Small ulcers that penetrate into the anterior chamber generally give rise to permanent adhesions of the iris to the corneal tissue at the point of perforation, and so produce **anterior synechia.** Large and dense maculae accompanied by such synechiae are known as **adherent leukomata**. Extensive perforating ulcers sometimes give rise to **prolapse of the iris** and **corneal staphyloma** (Art. 344).

Ulcers at the corneal margin as they heal sometimes exert traction on the adjacent conjunctiva, which thus comes to overlap the cornea, and forms what is known as a **pterygium**. In certain cases however a pterygium grows over the cornea without any antecedent ulceration. The origin of this peculiar affection is in many cases difficult to determine: it usually affects the interpalpebral region of the cornea, which is overlaid by a triangular or wing-shaped process of the conjunctiva, with its apex towards the corneal centre.

360. The numerous clinically distinguishable forms of keratitis may be divided into two groups, the circumscribed and the diffuse, the former being the commonest of corneal affections. Of circumscribed inflammations the most important is **phlyctenular keratitis**, also described as lymphatic or scrofulous keratitis, a very common eczematous affection that is often associated with

eczematous conjunctivitis. The foci of inflammation in the corneal affection vary quite as much in point of size as in the conjunctival form, though as a rule they are smaller and less prominent. They generally assume the form of slightly-raised greyish-white superficial elevations, about 0·5 to 1·5 millimetres in diameter, which soon become excavated in the centre by the disintegration of their apices. The larger the pustule the more deeply does it penetrate into the corneal tissue, the more distinct is the grey or yellowish turbidity of the cornea round about it due to infiltration with pus or leucocytes, and the deeper is the central pit, which may become excavated into a perforating ulcer. The eczematous pustules may be situated on the marginal as well as on the central parts of the cornea.

IWANOFF found that a newly-formed corneal pustule was composed of a dense aggregation of leucocytes, which pushed up the epithelium into a small eminence and extended to Bowman's membrane.

A peculiar variety of corneal eczema, known as **fascicular keratitis**, is distinguished by the formation of a 'wandering pustule.' In this affection a pustule near the margin is converted into a small ulcer, into which a few vessels extend from the limbus of the conjunctiva, while the opposite edge of the ulcer towards the corneal centre becomes converted by infiltration into a whitish crescentic rampart, which slowly advances across the cornea closely followed by the vessels. When the process continues for some time, the crescentic edge travels over a large part of the cornea, its concave side being throughout connected with the margin of the cornea by a leash of vessels lying in a shallow groove.

The repair of deep eczematous ulcers is always accompanied by the ingrowth of vessels from the nearest point of the corneal margin.

Febrile or **catarrhal herpes** is much rarer than eczema, and herpes zoster is still more infrequent. The former eruption consists of a row or cluster of slightly-raised vesicles, from 0·5 to 1·5 millimetres in width, and containing a clear watery liquid. The thin roof of the vesicle soon gives way, and an ulcer with ragged edges is formed, which in cases of moderate severity is remarkably and indeed characteristically slow in healing. The roof of the herpetic vesicle probably consists not only of epithelium, but of a superficial layer of corneal tissue also, for the depth of herpetic ulcers of some standing is apt to be very considerable (Fig. 554 *G*), and sometimes extends to the middle layers of the cornea. The floor and edges of the ulcer (*G J*) are infiltrated with leucocytes. When repair begins the epithelium (E_1) grows from the margin towards the centre, as in other corneal ulcers. The restoration of the proper corneal tissue is usually late in beginning, no doubt because the formation of new vessels in the floor of the ulcer is a very slow process.

In catarrhal herpes there is in general no herpetic eruption on the face, but **ophthalmic herpes zoster** is usually accompanied by an eruption of vesicles along the course of the trigeminal twig on the same side. The corneal vesicles are as clear as water, and soon rupture. The resulting ulcer is often very large, and its floor and edges generally undergo inflammatory infiltration more quickly and to a greater extent than in catarrhal herpes, with the result that hypopyon and iritis are frequently induced. The repair of the erosion, which in this affection also extends beyond the epithelial layer, is even slower than in catarrhal herpes.

Ophthalmic herpes zoster often leads merely to the production of large or small infiltrations, but neuroparalytic keratitis occasionally supervenes (Art. 361).

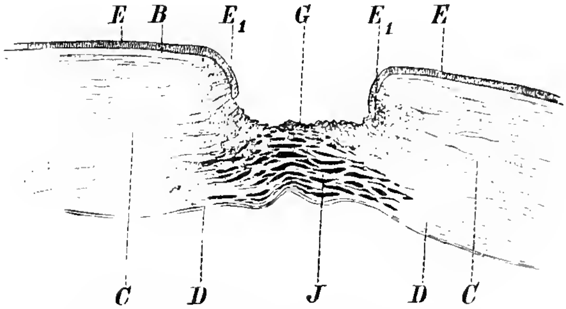

FIG. 554. CATARRHAL HERPES OF THE CORNEA.

(*Ulcer in process of repair, three weeks after the commencement of the eruption*: × 20)

C	cornea
B	Bowman's membrane
D	Descemet's membrane
E	corneal epithelium
E_1	epithelium lining the sides of the excavation
G	infiltrated floor of the ulcer
J	infiltration beneath the floor of the ulcer

In severe cases of herpes zoster, the affection of the trigeminal nerve is manifested not only in the skin and cornea, but also in the interior of the eye. SATTLER describes a case in which, in addition to a shallow corneal ulcer and iritis, inflammation of the choroid and ciliary body with infiltration of the vitreous was present. The gasserian ganglion and the ciliary ganglion were infiltrated with round-cells.

In rare instances **acne** attacks both the conjunctiva and the cornea, the papules readily breaking down and leaving small circular ulcers that persist for a long time (VON ARLT).

Concerning **variola** see Art. 358.

361. Various forms of keratitis that begin as circumscribed cellular infiltrations and afterwards spread both over the surface and into the deeper layers, and not infrequently result in extensive destruction of the corneal tissue, are due to **mycotic infection** by germs that have manifestly gained access through minute erosions of the corneal surface. This is the case with the inflammatory infiltrations that appear in the course of gonorrhoeal and

diphtherial conjunctivitis, and with the infiltrations of the central parts of the cornea that sometimes occur in blennorrhoea neonatorum, all of which tend to issue speedily in ulcerous disintegration. WEDL and BOCK found colonies of micrococci in a diphtherial ulcer of the cornea.

In the **corneal softening** (*keratomalacia*) of infants from two to four months old, who are the subjects of fatal marasmus from digestive disorder, it can be shown that large numbers of micrococci penetrate deeply into the corneal tissue. In this affection also the infiltration first appears in the exposed part of the cornea round a small epithelial erosion; as in blennorrhoea neonatorum, the infiltration is rapidly converted into an ulcer with a greyish-yellow floor and yellow edges, which tends steadily to increase in breadth and depth, and ends in perforation.

When the ulcer is actually formed (Fig. 555), the interstices of the cornea around it are in general densely packed with micrococci (Fig. 555 M M_1 and Fig. 556 M), and in some cases these may also be found at a considerable distance (Fig. 555 M) from the ulcer. The cellular or purulent infiltration (J) partly surrounds the ulcer and partly follows the colonies of micrococci, but always in such a manner that the space immediately around the latter is left free. FRAENKEL and FRANKE, like BAUMGARTEN, were able to discover only *Staphylococcus pyogenes aureus* in the corneal tissue, the bacillus associated with xerosis being absent.

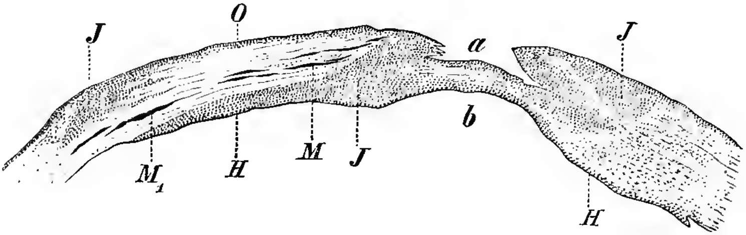

FIG. 555. MYCOTIC KERATITIS.
(*Keratomalacia infantum*)

a	corneal ulcer with undermined edges	*O*	anterior surface
b	forward bulging of the floor of the ulcer	*J*	cellular infiltration
H	posterior surface of the cornea	*M* and M_1	colonies of micrococci

Neuroparalytic keratitis, following paralysis of the trigeminus, is manifestly akin to mycotic keratitis. If in a young rabbit the trigeminus be divided within the skull, an opacity soon appears near the centre of the cornea (HAAB), the epithelium becoming necrotic and converted into a flaky yellowish mass, traversed by small fissures, while the cells of the underlying corneal tissue cease almost or altogether to stain with haematoxylin. At the same time the deeper layers of the cornea are infiltrated with leucocytes. As the necrotic epithelium and the superficial layers of

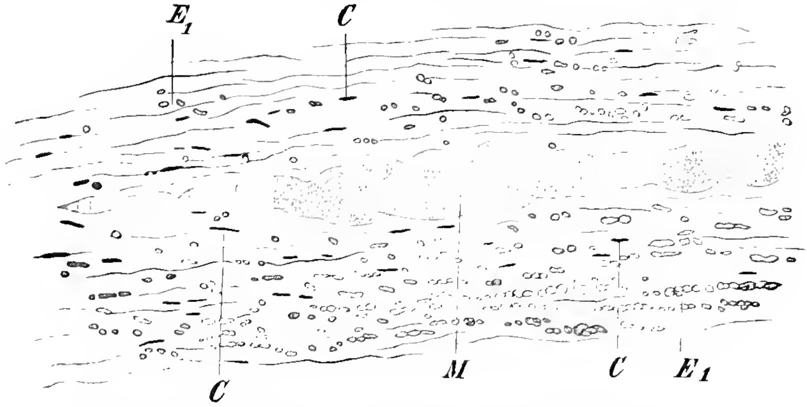

FIG. 556. COLONY OF MICROCOCCI IN CORNEAL SOFTENING.
(*From the point M in Fig. 555, under a higher magnifying power*)

C nuclei of corneal cells
E_1 pus-corpuscles, some of them near the posterior surface of the cornea
M mass of micrococci between the corneal lamellae

the cornea break down an ulcer is formed, whose floor is more or less completely covered over with masses of micrococci, which sometimes send out prolongations that penetrate between and loosen the fibrils of the cornea. The micrococci are small and aggregated into zoogloea. As they advance, the immigration of pus-corpuscles into the corneal tissue increases, and gives rise to dense infiltration of the margins of the shallow ulcer, which as it deepens is apt at length to perforate through the cornea. According to Gaule, section of the nerve at the gasserian ganglion is at once followed by definite tissue-changes, partly necrotic and partly proliferous, the latter affecting even the endothelium of Descemet's membrane. True inflammation does not make its appearance until after these changes have taken place.

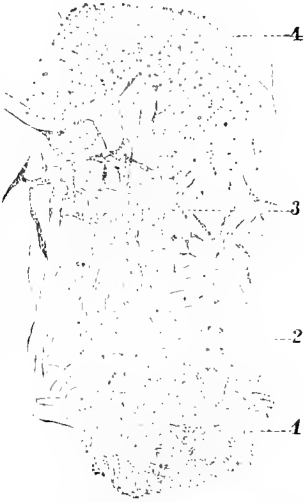

FIG. 557. MYCOTIC KERATITIS IN FACIAL ERYSIPELAS.
(*Preparation from Prof.* HORNER'S *collection; horizontal section;* × 70)

1 edge of the central infiltration
2 zone of necrotic disintegration
3 zone of micrococci
4 zone of marginal infiltration

Creeping ulcer (*ulcus serpens*), another form of ulcer affecting the interpalpebral part of the cornea, is also due to infection by micrococci. The tissue adjoining the ulcer exhibits a peculiar yellowish-grey infiltration of greater or less extent, often surrounding half the circumference the ulcer; and this infiltration spreads rapidly across the cornea, the ulceration following close upon it. SATTLER and others have found colonies of micrococci in the floor and edges of spreading ulcers of this kind.

In facial **erysipelas** also the cornea may be the seat of mycosis with ulceration. The floor of the erysipelatous ulcer is usually infiltrated with pus (Fig. 557, 1), and is surrounded by a zone (2) within which the corneal cells have perished. Still further outwards the interlamellar spaces are filled with micrococci (3) and the contiguous tissue infiltrated with cells (4) which have migrated from the corneal margin.

Rodent ulcer of the cornea is a peculiar and very rare superficial erosion, whose floor is slightly infiltrated and pervaded by capillary vessels. It has peculiar whitish somewhat abrupt and often undermined edges, enlarges slowly but steadily, and in the course of months removes evenly the surface layers of the cornea. The microscopical appearances presented by the lesion have not yet been fully described.

The so-called **dendritic keratitis** is identical with catarrhal herpes.

Circumscribed corneal inflammations due to tuberculosis, syphilis, and leprosy are very rare. MANZ has observed a number of small recent tubercles embedded in a subepithelial patch of cellular infiltration at the lower border of the cornea. ROY and ALVAREZ demonstrated the presence of tubercle-bacilli in a nodular corneal infiltration (*Rev. clin. d'oculistique* **1885**).

Keratomycosis aspergillina, a disease due to the settlement of *Aspergillus* on the cornea, and leading to the formation of large suppurating ulcers and hypopyon, has been hitherto but seldom observed (LEBER: *A. f. Ophth.* XXV **1879** and *Die Entstehung der Entzündung etc.* Leipzig **1891**; LIPPMANN: *Inaug. Diss.* Berlin **1882**).

362. The best example of diffuse corneal inflammation is **diffuse interstitial keratitis** (*keratitis profunda*, *parenchymatosa*, or *scrophulosa* of VON ARLT and MACKENZIE, the syphilitic keratitis of HUTCHINSON), a disease generally affecting both eyes and commonest in the children of syphilitic parents (HUTCHINSON). HORNER ascertained that hereditary syphilis was present in 64 per cent. of the cases, SAEMISCH in 62 per cent., and MICHEL in 55 per cent. FOURNIER also regards the inflammation as due to hereditary syphilis. A small proportion of the affected children exhibit signs of scrofula.

In the incipient stages of the disease very slight hyperaemia makes its appearance round the cornea, and a faint greyish turbidity is perceptible at a point on the corneal margin; this slowly enlarges and spreads over the cornea, while similar cloudy spots form at other marginal points and spread in like manner. At length the entire margin becomes clouded by the coalescence of these spots, and as the infiltration advances from all sides towards the centre of the cornea the turbidity ultimately becomes most dense at this point. The marginal turbidity gradually clears up again, though the clarification is often only partial, isolated *nebulae* made up of faint hazy spots lying in different layers of the cornea sometimes persisting for a long time. In the course of weeks or months the entire cornea usually regains its normal transparency, though at points where the infiltration has been very persistent or frequently recurrent permanent nebulae of varying degrees of intensity are apt to be left.

The surface of the cornea never undergoes ulceration, but over the infiltrated portions it loses its characteristic lustre and acquires the appearance of ground glass. In the course of the disease, and especially in its later stages, new vessels usually make their appearance in the cornea. The vessels are often very delicate, and almost invisible, but they traverse even the deeper layers in a radial direction. In other cases they run so closely together in the outer layers that the entire cornea assumes an intense greyish-red tint.

This form of keratitis is often accompanied by iritis, generally of the serous kind, and after the inflammation has run its course deep-seated complications are often discovered, such as opacities of the vitreous, posterior polar cataract, marginal choroiditis, and the like.

Similar diffuse infiltrations have been observed, although rarely, after intermittent fever (VON ARLT), after bruising without laceration of the cornea, in the neighbourhood of punctured or incised wounds and patches of scleritis, and lastly in the secondary stage of acquired syphilis. Keratitis of this kind is however rare as a syphilitic lesion, and the turbidity is usually from the outset somewhat cloudy and irregular, being made up of clusters of small faint specks.

It should moreover be noted that in severe cases of iritis a faint diffuse clouding of the cornea, due to the immigration of leucocytes, can very often be detected.

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HORNER: Phlyctenular keratitis and herpes of the cornea *Gerhardt's Handb. d. Kinderkrankh.* V Tübingen **1882**

HUTCHINSON, J.: Diffuse interstitial keratitis *Diseases of the eye and ear consequent on inherited syphilis* London **1863**

IWANOFF: Phlyctenular keratitis *Klin. Monatsbl.* **1869**

KEY and WALLIS: *V. A.* 60 **1874**

KRÜCKOW: *Klin. Monatsbl.* **1875**

LEBER: *Die Entstehung der Entzündung etc.* Leipzig **1891**
SATTLER: Herpes zoster *Wien. med. Presse* **1875**
SILVESTRI: *A. f. Ophth.* XXXVII **1891**
TALMA: *A. f. Ophth.* XVIII **1872**
WEDL and BOCK: *Pathologische Anatomie des Auges* Vienna **1886**

363. **Inflammation of the sclerotic.** The sclerotic is much less frequently attacked by inflammation than the cornea, and the affections of the part of it anterior to the equator of the eye-ball are the only inflammatory lesions of which we have any certain knowledge; indeed it is still questionable whether the posterior segment is susceptible of inflammation.

Scleritis may occur either alone (when it is described as simple or solitary scleritis, or **episcleritis**), or in association with inflammation of the cornea, iris, or choroid (kerato-scleritis or irido-scleritis). In simple scleritis there appears between the margin of the cornea and the equator, at a distance of from 3 to 7 millimetres from the former, a circumscribed hyperaemic patch which swells into a prominence several millimetres in width, and soon assumes a faint bluish-red colour. The highly-injected and often somewhat oedematous conjunctiva is stretched evenly over the crest of the prominence, which accordingly is not eroded as it would be in the case of a large eczematous pustule of somewhat similar appearance. The prominence after a time slowly subsides and disappears without leaving a trace, or its place is presently taken by a greyish discoloration of the sclerotic. The infiltration producing the swelling occasionally extends round the cornea, always however leaving behind it the characteristic greyish discoloration, which is due to attenuation of the sclerotic (*scleritis migrans*), until at length, when the disease has run its course, the entire circumcorneal zone has acquired a slaty-grey tint. In very chronic cases, again, the portion of the cornea adjacent to the initial scleritis may become implicated, a deep-seated diffuse wedge-shaped infiltration appearing in it, and sometimes extending far into its substance: this affection has been described as sclerosing keratitis. BAUMGARTEN however has ascertained that no true sclerosis of the corneal fibrils takes place, the morbid change consisting in dense cellular infiltration and fatty degeneration of the corneal tissue.

Scleritis is liable to accompany diffuse interstitial keratitis, chronic inflammation of the iris (especially serous iritis), and chronic choroiditis. In the latter case the scleritis is generally of a diffuse character, and occasionally leads to ectasis of the anterior segment (sclerotic staphyloma). Microscopical examination (BAUMGARTEN, UHTHOFF) shows that in scleritis the tissue is thickly infiltrated with leucocytes, especially around the vessels, and that the lymphatics are sometimes widely dilated.

Tuberculosis of the sclerotic has but rarely been observed. Inflammatory lesions due to **syphilis** are somewhat more frequently met with, gummata being the commonest: they give rise to prominences that are larger and yellower than those characteristic of ordinary scleritis.

References on Scleritis.

BAUMGARTEN: *A. f. Ophth.* XXII 1876
UHTHOFF: *A. f. Ophth.* XXIX 1883

364. All severe or protracted inflammations of any one of the three divisions of the **uveal tract** (iris, ciliary body, and choroid) are apt not only to involve the others, but also to extend to the contiguous structures that lie outside this region.

Inflammation of the **iris**, or **iritis**, is manifested by the following symptoms: first, the anterior ciliary vessels and those of the conjunctival limbus with which they communicate become hyperaemic, a condition spoken of as ciliary or circumcorneal injection, and appearing as a faint bluish-red zone from 3 to 6 millimetres wide immediately around the cornea. Then the iris loses its lustre, owing partly to a slight turbidity of the aqueous humour, and its colour changes and becomes redder from local diffuse hyperaemia, but no large injected vessels are usually visible. In the next place adhesions of the margin of the pupil to the anterior capsule of the lens are set up, and constitute a cardinal symptom of the disease. These adhesions of the iris, which are known as posterior synechiae, are sometimes very slender, and when the pupil dilates look like sharp projections from its edge that are firmly adherent to the capsule; in other cases they are broad and connect to the capsule a considerable arc of the pupil, or it may be even its entire circumference (annular synechia or occlusion of the pupil). In severe cases the iris is visibly thickened and softened, the turbidity of the aqueous humour becomes denser, hypopyon may be formed at the lower part of the anterior chamber by the accumulation therein of purulent liquid, and the entire area of the pupil may be occupied by a membranous exudation (*obturatio pupillae*).

This last-named variety of inflammation is also called **plastic iritis**, to distinguish it from another form, **serous iritis**, in which the visible changes in the iris are less conspicuous, though punctiform deposits on the posterior surface of the cornea indicate the supervention of exudative inflammation. In serous iritis the circumcorneal injection may be almost entirely absent, and there may be no discoloration of the iris and no synechiae. The deposits are greyish or greyish-brown, and generally appear as minute points scattered over the lower part of Descemet's membrane. When they are larger (up to the size of a pin's head) they have a greyish-white colour, and are placed nearer the margin of the cornea. It was formerly thought that these deposits were situated within the substance of the cornea, and the affection was therefore erroneously termed *keratitis punctata*. Serous iritis is apt to be somewhat chronic, and is generally but one part of a more general inflammation affecting the entire uveal region: signs of inflammation in the ciliary body (cyclitis) and in the choroid are thus usually discernible in the later stages, the affection then passing into **irido-choroiditis**.

The plastic and serous forms cannot be very sharply distinguished: for on the one hand the inflammatory infiltration of the iris in serous iritis is really more abundant than might be inferred from the gross appearances; and on the other hand while chronic serous iritis is often accompanied by synechiae and even by adhesion of the entire posterior surface of the iris to the capsule, in plastic iritis exudative deposits on Descemet's membrane are not uncommon.

In fibrino-purulent plastic iritis, which is readily induced by the introduction of irritant substances into the anterior chamber, the endothelial film covering the anterior surface of the iris is, according to MICHEL, detached from the subjacent tissue by a fibrino-cellular exudation. A fibrino-purulent effusion is poured out between the posterior surface of the iris and the capsule of the lens, and in front of the endothelial film into the anterior chamber. The connective tissue of the iris is swollen, its vessels are over-distended, and it often exhibits haemorrhagic extravasations and more or less abundant diffuse cellular infiltration around the vessels. The border of the iris is agglutinated to the mass of fibrinous exudation within the pupillary area.

According to KNIES, the punctiform greyish deposits on the posterior surface of the cornea in serous iritis consist of small aggregations of round-cells that have migrated from the iris, mingled with detritus and pigment-granules. Underneath the larger specks the endothelial lining of Descemet's membrane is destroyed, but underneath the smaller ones it appears intact. The iris is densely infiltrated with cells, the infiltration being more abundant towards the anterior surface, and being in places aggregated into circumscribed thickenings. The ciliary body and the choroid are similarly infiltrated. The implication of the choroid in the inflammation appears to be the ordinary result not only in this form of iritis, but also in the fibrino-purulent variety. SATTLER at any rate observed that immigrant round-cells were almost invariably present in the chorio-capillaris of eyes that had suffered from iritis and irido-cyclitis.

365. **Syphilitic iritis** is very common and generally affects both eyes. It differs in no appreciable respect from the plastic iritis associated with injury, rheumatism, etc., although it sometimes assumes a transitional form between the plastic and the serous varieties. Nodes varying from 2 to 6 millimetres in diameter are not infrequently formed in the tissue of the iris, and are usually described as gummata. According to MICHEL and FUCHS, however, inflammations of apparently simple type are also accompanied by the formation of similar nodules. The yellowish-red syphilitic nodules that are externally visible about the pupillary margin are made up of closely-packed young connective-tissue cells permeated by turgid vessels. The cells are small, with rounded highly-refractive nuclei and scanty protoplasm. COLBERG observed that the cells of the adventitia of the vessels were also proliferous.

Fuchs met with giant-cells in small syphilitic nodules of the iris, and around these as in the rest of the iris the vessels exhibited signs of syphilitic disease.

Neumann has described a gumma that included portions of the iris, ciliary body, sclerotic, choroid, and retina, the section of which appeared in some parts white and marrowy, and in other parts mucoid and colourless; it consisted of soft cellular tissue permeated by a close network of capillary vessels. The apparently unaffected portions of the iris and choroid contained large aggregations of cells extending far beyond the limits of the visible infiltration, and confined chiefly to the course of the vessels.

Tuberculous iritis is a rare lesion, generally affecting one eye only, and characterised by a slowly-advancing and long-continued eruption of tuberculous nodules in the tissue of the iris. The affection has long been recognised, but was generally described as **granuloma**. It sometimes begins with the symptoms of serous iritis; but the iris soon appears beset with small greyish tubercles, usually seated about its ciliary attachment and in the canal of Fontana, though they are occasionally to be detected on the flat surface also. As they increase in size and number they assume the appearance of an irregular greyish-red vascular growth, which gradually fills up the anterior chamber, and is often accompanied by turbidity and vascularity of the cornea. The process may at this stage be arrested, and the growth then gradually diminishes in size and disappears altogether in the course of a few months; in other cases new tubercles continue to be formed, and as they spread destroy both the ciliary body and the contiguous part of the sclerotic, until at length a caseous protuberance or ridge appears at the corneal margin, and this is soon followed by wasting and general disintegration of the eye-ball (*phthisis bulbi*). The choroid often remains entirely free from tubercles. It is worthy of note that, in most of the cases hitherto observed, the initial seat of the tuberculous lesion was in the lower half of the iris.

References on Iritis.

Adams: Unusual forms *Ophth. Hosp. Reports* x London **1881**
Fuchs: Gumma of the iris *A. f. Ophth.* xxx **1884**
Graefe and Colberg: Gumma of the iris *A. f. Ophth.* viii **1861**
Higgens: *Guy's Hosp. Reports* xix London **1874**
Knies: Serous iritis *A. f. Augenheilk.* ix **1880**
Michel: *A. f. Ophth.* xxvii **1881**
Neumann: Gumma of the ciliary region *A. f. Ophth.* xiii **1867**
Sattler: *A. f. Ophth.* xxii **1876**

References on Tuberculous Iritis.

Brailey and Edmunds: *Trans. Ophth. Soc.* ii London **1882**
Haab: *A. f. Ophth.* xxv **1879**
Haensell: *ibidem* **1879**

KNAPP: *Helmholtz's Festschrift* Stuttgart **1891**
KÖSTER: *Cent. f. med. Wiss.* **1873**
MANFREDI: *Annali di ottalm.* IV **1874**
PRUNEDA, COSTA: *A. f. Ophth.* XXVI **1880**
SAMELSOHN: *Berl. klin. Woch.* **1879**
WEISS: *A. f. Ophth.* XXIII **1877**
WOLFE: *Clinical demonstrations* London **1884**

366. Inflammation of the **ciliary body**, or **cyclitis**, involves chiefly the non-muscular parts of this organ, in other words the vascular ciliary processes and the plane portion as far as the *ora serrata* of the retina. It is frequently combined with inflammation of the choroid and iris. The first visible signs are a slight turbidity of the aqueous humour and the anterior portion of the vitreous, the presence of deposits on the posterior surface of the cornea, and some exudation within the region of the pupil. This is soon followed by adhesion of the entire posterior surface of the iris to the capsule of the lens and by retraction of the ciliary zone of the iris, whereby the peripheral portion of the anterior chamber becomes deepened. The inflammatory exudation poured out from the ciliary processes into the spaces between the iris and lens, and between the attachment of the iris and the ciliary processes (fornix of the posterior chamber), forms deposits that gradually become organised into cicatricial tissue, by the contraction of which the iris is drawn backwards. The exudations in front of and behind the lens may in like manner become organised and contract, dragging the ciliary body forcibly away from the sclerotic towards the axis of the eye-ball. The internal changes in the vitreous that invariably follow upon cyclitis, and are manifested by turbidity due to cellular and fibrinous exudations, by the appearance of filmy shreds and membranes, and by general contraction, usually bring about complete detachment of the retina and cataractous opacity and shrinking of the lens. When the inflammation is intense and the exudation becomes purulent, hypopyon is sometimes formed in the anterior chamber, though the iris may as yet be but slightly affected. Purulent cyclitis often leads to inflammation of the entire uveal tract and to **panophthalmitis**, while fibrinous cyclitis terminates in slow atrophy of the globe (*phthisis bulbi*), a characteristic symptom of this latter termination being intermittent or persistent diminution of the intra-ocular pressure.

The cause of cyclitis, other than inflammation extending directly to the ciliary body from the iris or choroid, is generally traumatic injury from wounds or the intrusion of foreign bodies. It sometimes happens that a wound of the sclero-corneal junction excites no ciliary inflammation until it reaches the stage of cicatrisation, when the scar contracts and presses on the ciliary body, or yielding to pressure bulges outwards and drags with it both the ciliary body and the iris.

Wounds sometimes lead not only to inflammation of the ciliary body that has been injured, but also to cyclitis in the other eye, a condition described as **sympathetic ophthalmitis.** This usually occurs only in cases of cyclitis or irido-cyclitis that are associated with perforation of the ocular capsule by punctured or incised wounds, rupture, foreign bodies, or ulcers, and never results from spontaneous or idiopathic inflammation in the first eye. The second eye may be attacked by sympathetic inflammation within three weeks of the injury to the first, or not till after the lapse of twenty years. In the latter case, active inflammatory changes are generally discoverable in the exciting eye, though a long interval of quiescence may have intervened. Even an atrophic and shrunken eye is capable of exciting sympathetic inflammation in the other.

The manner in which the inflammation extends to the other eye is not yet clearly understood. The hypothesis that it is transmitted by way of the ciliary nerves has of late been given up by most observers, who are now inclined to adopt the view originally put forward by MACKENZIE that the optic nerve is the channel of transmission. The researches of DEUTSCHMANN (*A. f. Ophth.* XXX **1884** and *Ueber die Ophthalmia migratoria* Hamburg **1889**) and of LEBER show that sympathetic inflammation spreads along the optic nerve in the form of neuritis and perineuritis. The former writer believes that the irritation is propagated by the action of micro-organisms. GIFFORD (*A. f. Augenheilk.* XVII **1887**) showed that in rabbits anthrax-bacilli can pass from the vitreous of the first eye along the larger vessels of the optic nerve, then outside the dural sheath of the nerve from the orbit into the cranial cavity, and multiplying therein pass back again through the subcapsular space and the sclerotic into the suprachoroidal lymph-space of the second eye.

367. **Choroiditis,** or inflammation of the **choroid,** often extends to the iris and ciliary body (irido-choroiditis) or to the retina (chorio-retinitis), and in any case inflammatory products from the choroid are very apt to make their way directly into the retina or vitreous.

In acute choroiditis, according to SATTLER, it is first the unpigmented layer (a delicate elastic network traversed by small arteries and veins) lying external to the chorio-capillaris, and afterwards the chorio-capillaris itself, that are the chief seats of cellular infiltration, the external pigmented layers being less affected. As the infiltration becomes more abundant the line of demarcation between the chorio-capillaris and the infiltrated unpigmented layer, which is often also studded with haemorrhagic extravasations, becomes effaced; the succeeding pigmented layers (the stratum of large vessels and the suprachoroid) are usually much less densely infiltrated with cells, though they are more apt to be forced asunder by fibrinous exudations and beset with small ecchymoses. In acute suppurative choroiditis, hyaline deposits interspersed with pus-corpuscles are not infrequently found upon the inner surface of the hyaline membrane next the retina, whereby the pigmentary epithelium of the latter is broken through and destroyed.

Acute suppurative choroiditis is induced by infection, and accordingly is liable to supervene on septic wounds or ulcers of the cornea and sclerotic, and also in cases of septic embolism and cerebro-spinal meningitis. It is accompanied by rapidly-increasing chemosis of the ocular conjunctiva, slight exudation in the pupillary region, and hypopyon, these symptoms being speedily followed by the appearance of a greenish-grey reflexion from the fundus of the eye, due to the accumulation of pus in the vitreous. In cerebro-spinal meningitis the choroidal inflammation reaches at this stage its climax, its subsequent course resembling that of cyclitis; but septic inflammation of the uveal tract usually spreads rapidly over the whole eye, being manifested by oedematous or purulent inflammation of Tenon's capsule, and by immobility and protrusion of the eye-ball, in a word by **panophthalmitis**, the cornea at the same time becoming infiltrated with pus. The pus may thereupon burst through the capsule of Tenon, or the eye-ball may simply become wasted and shrivelled.

Metastatic ophthalmitis is set up by septic or micro-parasitic emboli, and makes its appearance in the course of general pyaemia: inflammation of one or both eyes is in some cases indeed the only pyaemic metastasis in the whole body. Any septic focus, from a whitlow to a puerperal metritis (the latter being a very common source), may furnish the matter for such an embolus. Mycotic endocarditis, with valvular colonies of micrococci, is often an intermediate or a primary source of infection. HEIBERG, HOSCH, and also WEDL and BOCK, detected masses of zoogloea in the vessels of the choroid and retina; MICHEL observed them in the iris.

Most of the chronic inflammations of the choroid are of the nature of **disseminated choroiditis** and **chorio-retinitis**. In these affections circumscribed inflammatory patches appear in the choroid (the iris being usually exempt), and are at first discrete and confined either to the margin or to the central parts of the membrane. As they become more numerous, by the formation at intervals of new patches in various places, they tend here and there to coalesce into larger ones. At first the patches are yellowish-red in colour and their contours are not very distinct, but as they enlarge their colour changes through yellow to white, and within the pale-tinted, round, oval, or irregularly-shaped spots so produced appear islands of pigment of various sizes (Fig. 558). Often too the edges of the patches are bordered with pigment.

From the outset rounded or irregular pigment-spots of a deep black colour are occasionally interspersed among the pale-yellow or white patches; these also enlarge slowly and are often very numerous, sometimes indeed outnumbering the others. A variety of the affection, generally most marked in the posterior part of the choroid, and described by FÖRSTER as areolar choroiditis, is characterised by the appearance of pale centres in the slowly-enlarging spots of pigment. The entire choroid occasionally becomes affected in this way, though at times only one or two isolated patches are produced. The fundus of the eye may escape and only the peripheral parts suffer, or the reverse. The latter condition is known as posterior choroiditis, and is usually associated with grave retinal inflammation: indeed in every case of active choroiditis the retinal vessels are highly distended, and as a rule capillary hyperaemia of the optic papilla is also apparent.

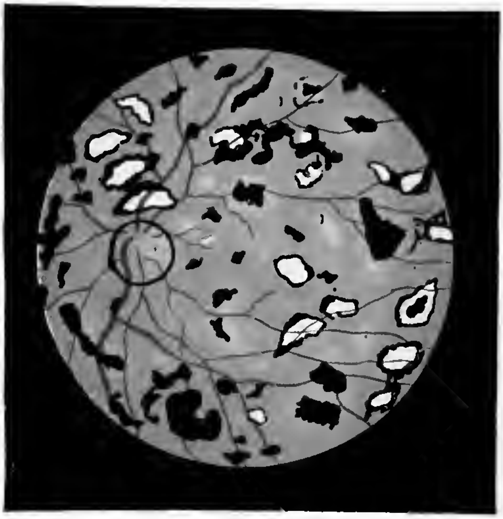

FIG. 558. DISSEMINATED CHOROIDITIS.
(*Ophthalmoscopic appearance of the fundus*)

The patches, so long as they are small and covered over with uninjured pigmentary epithelium, consist of a vascular aggregation of closely-packed spherical or fusiform leucocytes. In the larger patches amorphous fibrinous exudations are interposed between the cells, and the pigmentary epithelium disappears as the patches become adherent to the retina; sometimes however the epithelial cells lose their colour and become flattened out (Duke CARL THEODOR of Bavaria). The nodules at times vanish again completely (VON WECKER), but more frequently they give rise to circumscribed atrophic patches, to which the retina is closely adherent (Fig. 559 *V*). This latter condition is dependent

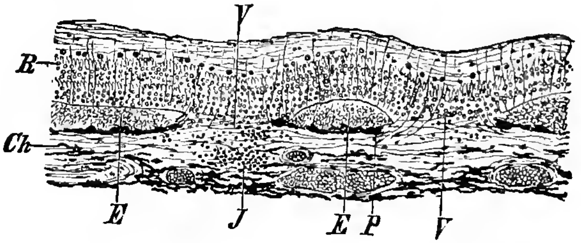

FIG. 559. LATER STAGE OF DISSEMINATED CHOROIDITIS (× 20).

Ch	choroid
R	retina, in places (*V*) adherent to the choroid, the bacillary layer and the pigmentary epithelium being absent
P	proliferous pigmentary epithelium
J	aggregation of leucocytes in the choroid
E	amorphous exudation between choroid and retina

on the fibrous metaplasia that takes place in the older choroiditic patches and the simultaneous disappearance of the hyaline membrane, which is followed by the proliferous ingrowth of the radial fibres of the retina into the choroid, and by the disintegration of the bacillary and external nuclear layers of the retina, at these points. The pigmentary epithelium, on the other hand, often undergoes rapid proliferation, and large aggregations of pigment are thus produced (Fig. 559 *P*).

The choroid may be totally or partially destroyed at the points where the earlier nodular infiltrations were situated, leaving nothing but a little fibrous tissue containing few or no blood-vessels: such spots have a glaring white appearance when viewed with the ophthalmoscope. The pigmented patches, often so numerous, are derived partly from the pigmentary epithelium, and partly from the choroidal pigment.

Duke CARL THEODOR of Bavaria showed that in albuminuria the arterioles of the choroid were liable to be extensively diseased.

References on Suppurative Choroiditis.

HEIBERG: *Cent. f. med. Wiss.* **1874**
HIRSCHBERG: *A. f. Augenheilk.* IX **1880**
HOSCH: *A. f. Ophth.* XXVI **1880**
KNAPP: *A. f. Ophth.* XIII **1867**
MICHEL: *A. f. Ophth.* XXVII **1881**
ROTH: *D. Z. f. Chir.* I **1872**
SATTLER: *A. f. Ophth.* XXII **1876**
WEDL and BOCK: *Path. Anatomie d. Auges* Vienna **1888**

References on Disseminated and Syphilitic Choroiditis.

Duke CARL THEODOR of Bavaria: *A. f. Ophth.* XXV **1879**; *Ein Beitrag zur pathol. Anatomie des Auges bei Nierenleiden* **1887**
IWANOFF and VON WECKER: *Graefe and Saemisch's Handbuch* IV **1876**
NETTLESHIP: Syphilitic choroiditis *Ophth. Hosp. Reports* II London **1887**
SCHÖN: *Klin. Monatsbl.* XIII **1875**

368. **Syphilitic inflammation** is much less frequent in the choroid than in the iris; but large and small patches of syphilitic choroiditis scattered over the equatorial region, and very similar to those described under disseminated choroiditis, are sometimes observed. Another form of syphilitic inflammation is manifested merely by diffuse dusty-looking haziness of the vitreous, and particularly of its axial portions, whereby the disc and the fundus about it are rendered indistinct. Another somewhat rare form leads to dense and wide-spread infiltration of the choroid and retina about the posterior pole, resulting in extreme fibroid atrophy of these parts. Minuter anatomical investigation of these syphilitic inflammations of the choroid is much to be desired.

Tuberculosis makes its appearance in the choroid either as an acute miliary eruption or as a chronic affection resulting in the formation of agglomerate nodes: the latter form is rare. The miliary form is usually associated with general miliary tuberculosis; according to COHNHEIM, indeed, miliary tubercles are found in the choroid in all cases of the general eruption, while LITTEN estimates the proportion at 75 per cent. The tubercles may be situated in any part of the choroid. Their number may be as few as 3 to 6, or as many as 50 to 60, and they vary in size from 0·4 to 1·5 millimetres. They lie beneath the chorio-capillaris (MANZ). The overlying retina is not appreciably altered, and is but slightly raised by the nodules. The vitreous shows quite as little change, while the ciliary body and the iris rarely contain miliary tubercles. Choroidal tubercles possess the characteristic structure, the parts of the choroid surrounding them being infiltrated with leucocytes.

Chronic tuberculosis of the choroid, associated with the formation of large agglomerate tubercles, was first discovered by A. VON GRAEFE on examining a pig's eye with the microscope. In the human choroid tumour-like growths are formed, whose centres become caseous. As they grow the sclerotic may be broken through, and the disease then extends beyond it. The ciliary body is sometimes the seat of a tuberculous node (NEESE).

References on Miliary Tuberculosis of the Choroid.

BRÜCKNER: *A. f. Ophth.* XXVI **1880**
BUSCH: *V. A.* 36 **1866**
COHNHEIM: *V. A.* 39 **1867**
FRÄNKEL: *Jahrb. f. Kinderheilk.* II **1868**
VON GRAEFE and LEBER: *A. f. Ophth.* XIV **1868**
LITTEN: *Volkmann's klin. Vorträge* no. 119 Leipzig **1877**
MANZ: *A. f. Ophth.* IV **1858** and IX **1863**
PERLS: *A. f. Ophth.* XIX **1872**

References on Chronic Tuberculosis of the Choroid.

VON GRAEFE: *A. f. Ophth.* II **1856**
HAAB: *Helmholtz's Festschrift* Stuttgart **1891**
MANZ: *Klin. Monatsbl.* XIX **1881**
NEESE: *A. f. Augenheilk.* XVI **1886**
WAGENMANN: *A. f. Ophth.* XXXIV **1888**
WEISS: *A. f. Ophth.* XXIII **1877**

369. The most striking form of **retinitis** is the purulent inflammation of the retina resulting from septic infection, whether the exciting cause gains access to the eye-ball by way of the circulation (metastatic retinitis) or through a wound or ulcer. The inflammation may spread rapidly from the retina to the choroid, or be limited to the retina alone, at least for a time: or again, as is most frequently the case in metastatic retinitis, infective emboli may make their way into the choroid and the retina, and excite inflammation of both membranes simultaneously. In all cases the process tends to extend to the whole of the globe and so give rise to panophthalmitis, when, as usually happens the entire retina is

rapidly destroyed by suppuration, and thick purulent deposits are formed on its inner surface.

In septic retinitis numerous haemorrhages first make their appearance in the retina, these being sometimes followed by purulent infiltration. HERRNHEISER noted the presence of septic retinitis in 30 per cent. of patients suffering from septic pyaemia. He moreover observed a form of the disease in which retinal haemorrhages were the only evidence of the general septic infection of the system. In such cases the retina presented no symptoms of inflammation and no indications of the presence of microbes. These latter (*Staphylococcus* and *Streptococcus pyogenes*) were however detected in many cases of metastatic retinitis attended by severe inflammatory symptoms, and in such cases the other parts of the eye (in particular the choroid and iris) were also involved.

When the inflammation runs a less violent course (as for instance in cases resulting from suppurative kerato-iritis, wounds, etc.) the purulent infiltration first spreads along the layer of nerve-fibres, and afterwards extends to the layer of ganglion-cells and other layers. The retinal tissue becomes cloudy and thickened, from infiltration with finely-granular and fibrinous exudations, interspersed with extravasated blood and fat-granule cells. In the later stages of the process the radial fibres undergo hypertrophy, with multiplication of their nuclei, and become elongated in the direction of the choroid, the bacillary layer having usually perished soon after the onset of the attack.

The chronic inflammation known as **albuminuric retinitis,** which is associated with Bright's disease, is of much more frequent occurrence, and generally involves the optic papilla also; it is attended by peculiar degenerative lesions, and these with the inflammatory changes give the affection a distinctive character. Around the optic disc, which is usually hyperaemic, swollen, and blurred, appear a number of radiating linear or rounded haemorrhages, interspersed with large and small white irregularly-shaped spots, which often coalesce into broad patches encircling the papilla. The retinal vessels, and particularly the veins, are abnormally distended and tortuous. In the neighbourhood of the yellow spot white specks are generally apparent, and are often so arranged as to form a characteristic stellate figure.

The changes just described are in general present in both eyes, though usually in different degrees of intensity.

The white spots correspond sometimes to dense aggregations of fat-granule cells lying chiefly in and between the nuclear layers, in other cases to clusters of clear translucent spherules and droplets, and in others again to colloid particles and flakes that resemble fibrinous coagula. The spherules and flakes are usually situated in the internuclear stratum, and are probably derived from extravasated blood. Swollen pyriform and spindle-shaped nerve-fibres and groups of bodies resembling ganglion-cells are also present; these according to H. MÜLLER originate from thickened nerve-fibres. The white spots forming the stellate figure in the macular region are due to fatty degeneration of the inner ends of the radial fibres. As regards inflammatory changes proper, we meet with numbers of leucocytes (Fig. 560 *L*), especially along the course of the vessels, fibrinous exudations in the interstices of the retinal tissue (*F*), and hyperplasia of the neuroglia. The morbid condition of the vascular system is manifested by wide-spread arteritis (Duke CARL THEODOR of Bavaria), sclerosis, and thickening of the coats of the smaller arteries (*A*) and capillaries. To the alterations in the walls of the vessels are no doubt due the numerous haemorrhagic extravasations (*B*) that take place within the retina. In the layer of nerve-fibres they are linear, in the deeper layers more rounded in form.

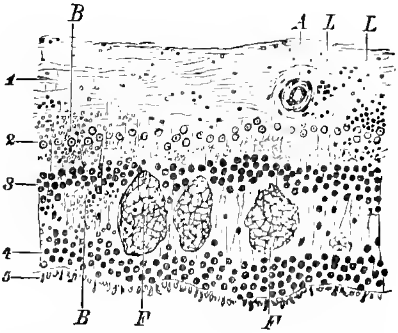

FIG. 560. ALBUMINURIC RETINITIS (× 70).

1	layer of nerve-fibres
2	layer of ganglion-cells
3	inner nuclear layer
4	outer nuclear layer
5	rods and cones (bacillary layer)
A	sclerotic artery
B	haemorrhage (red corpuscles)
F	fibrinous exudation in the internuclear layer
L	cluster of leucocytes

In the optic nerve the changes observed are cellular infiltration, hyperplasia of the interstitial connective tissue, and patches of grey degeneration.

In **diabetes** changes in the retina similar to those accompanying disease of the kidneys are met with, though they are less common. Simple retinal haemorrhage is however often observed.

The affection known as chronic **diffuse retinitis** is generally consecutive to inflammation of the uveal tract (cyclitis and irido-choroiditis) and is most marked in the inner layers of the retina, where its presence is manifested first by diffuse cellular infiltration and afterwards by interstitial fibrous hyperplasia. The radial fibres become thickened and elongated as the retina grows thicker, while the retinal connective tissue as a whole and the adventitia of the vessels become hyperplastic. The overgrowth of the radial fibres often extends beyond the retina, and forms a layer of reticular connective tissue of some thickness on the side next the vitreous. The nerve-elements on the other hand become scanty and atrophic, the layers of nerve-fibres and of ganglion-cells being the most affected, the bacillary layer the least. In particular instances the last-named layer undergoes a peculiar form of hypertrophy, which generally accompanies detachment of the retina from retinitis or other cause. The rods and cones, both in their outer and in their inner segments, become elongated to thrice their normal length and irregularly thickened, sometimes assuming the form of fibrillar or finely-striated agglutinate masses of a rounded shape and of considerable size.

Closely allied to this form of retinal inflammation is that described as **disseminated retinitis** of the external layers, which is observed in cases of disseminated chorio-retinitis. It is often difficult in these cases to decide whether the retinal affection is simply a secondary effect of the choroiditis, or of independent origin. Thin structureless films of exudation are intercalated between the choroid and the retina, the pigmentary epithelium being here and there destroyed and the rods and cones broken down (Fig. 559 *E*); in other cases the retinal pigment becomes in places proliferous (*P*), and so gives rise to the pigment-spots on the fundus seen with the ophthalmoscope. Presently the connective tissue of the outer retinal layers undergoes hyperplasia and grows out towards the choroid, the fibroid elevations so produced containing normal or disintegrated fragments of the bacillary layer, masses of pigment derived from the proliferous pigmentary epithelium, and colloid excrescences starting from the vitreous lamina of the choroid and either embedded in pigment or isolated. The pigment occasionally intrudes into the retina and there forms irregular deposits made up of granular masses. In the end the fibroid degeneration and pigmentation may extend to the inner layers of the retina, in which case the deposits of pigment appear to follow the course of the retinal vessels (Fig. 561).

In the form of retinitis just described the pigmentation is a secondary effect. There is however another affection in which pigmentation of the retina is from the outset the essential symptom, and which is accordingly known as **retinitis pigmentosa** (Fig. 561), though whether it is really an inflammatory process seems to be still doubtful. It is in general unaccompanied by any symptoms of inflammation, and might therefore be more appropriately described as a primary **pigmentary degeneration** of the retina. The ophthalmoscopic appearances characteristic of the affection are—contraction of the retinal vessels, especially of the arteries, faint yellowish-white (atrophic) discoloration of the optic papilla, whose outlines generally remain sharp, and last and most important a peculiar distribution of irregular deep-black pigmented patches about the periphery of the fundus over a zone lying between the posterior pole and the equator of the globe (Fig. 561). The pigment-spots are sometimes few in number and of small size, sometimes numerous and large. Some of them appear to surround the vessels or to lie in the line of continuation of these as they gradually disappear. In comparison with the rounded blotches characteristic of disseminated choroiditis these spots are slender and jagged like bone-corpuscles; only where they are very numerous and run together do they give rise to the appearance of large deep-black patches enclosing minute rounded lacunae. Haemorrhages and pale spots due to infiltration of the retina or atrophy of the choroid are never present. The disease always affects both eyes, and is sometimes hereditary.

The textural changes observed in the later stages of the affection include extreme fibrous hyperplasia of the retinal stroma, hyaline thickening of the coats of the vessels with obliteration of their smaller branches, and atrophy of the pigmentary epithelium, with the production of deeply-pigmented epithelial cells and intrusion of pigment into the retina, where it is deposited mainly in the sheaths of the vessels. All the nerve-elements of the retina, with the exception of the layer of nerve-fibres, are gradually destroyed. Colloid excrescences on the vitreous lamina are usually numerous in these cases. Wide-spread sclerosis of the vessels has moreover been detected in the uveal region, and particularly in the choroidal portion of it (WAGENMANN).

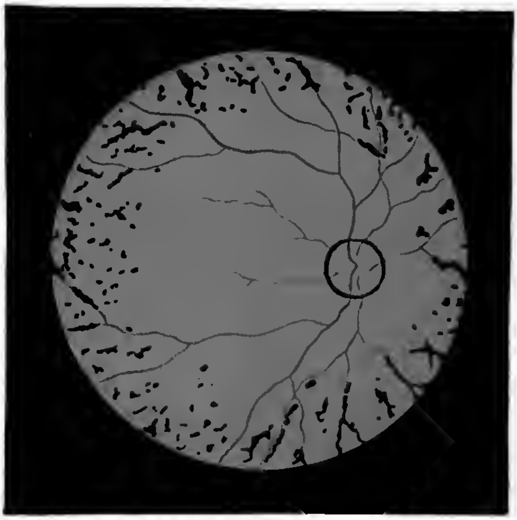

FIG. 561. RETINITIS PIGMENTOSA.
(Ophthalmoscopic appearance of the fundus of the eye)

Syphilitic retinitis, a not very common affection, appears in one or both eyes in two different forms: first, as a diffuse retinitis, similar to that just described, and secondly (but very rarely) as recurrent central retinitis. No histological examination of the latter form has as yet been recorded. With the ophthalmoscope a greyish opacity is observed in the fundus about the yellow spot.

Tuberculosis attacks the retina only in extremely exceptional cases.

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Landolt: *ibidem* xviii **1871**
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Pope: *Ophth. Hosp. Rep.* iv **1865**
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370. **Optic neuritis.** Inflammation may attack either the intra-ocular extremity of the optic nerve, or its trunk outside the globe (retrobulbar neuritis), or both together. Inflammation of the papilla, or **papillitis**, of greater or less intensity accompanies all the graver forms of retinitis (neuro-retinitis or papillo-retinitis). This association is well exemplified in the case of albuminuric retinitis. Papillitis may however arise without involving the retina, as when the inflammation spreads to the papilla from the trunk of the optic nerve or from the brain (descending optic neuritis), or when an intracranial tumour forces the subarachnoid liquid into the arachnoid sheath of the retrobulbar portion of the uninflamed optic nerve. In the latter condition the papilla often becomes rapidly and intensely swollen, the retinal veins showing signs of extreme engorgement, such as marked tortuosity and haemorrhagic extravasation: this form of papillitis is accordingly described, from its ophthalmoscopic appearance, as **choked disc.**

Papillitis is indicated ophthalmoscopically by the blurred outline of the optic disc, the papilla itself being cloudy, hyperaemic, and swollen, especially on the nasal side. The retinal veins are engorged, and the circumpapillary zone of the retina is somewhat hazy. In the severer forms of optic neuritis the swelling of the papilla is exceedingly marked. The increasing opacity of the tissues due to infiltration causes the vessels to become more and more overlaid and indistinct, especially at the edges of the disc, radiating linear haemorrhages make their appearance in the cloudy greyish-red fundus, and the entire disc is often so indistinct that its form can only with difficulty be recognised.

In choked disc the swelling and protrusion of the papilla and the engorgement of the retinal veins are more prominent than the opacity due to infiltration; but it is very often impossible to decide from the ophthalmoscopic appearances alone whether the papillitis is due to albuminuria, intracranial tumour, or descending neuritis. Even on microscopical examination the same tissue-changes are apparent in all three cases; but certain authorities lay stress on the fact that in choked disc the initial swelling of the papilla is due to oedema alone. Later on however the disc shows signs of inflammation also, namely cellular infiltration of the tissues, especially along the course of the vessels, varicose enlargements of the nerve-fibres, small extravasations of blood, and structureless finely-granular exudations between the strands of nerve-fibres. A few fat-granule cells and corpora amylacea are occasionally present. By and by fibrous hyperplasia and atrophy of the nerve-elements supervene, until finally a level or it may be slightly-concave fibroid disc occupies the place of the papilla; and when examined with the ophthalmoscope its dead-white colour, the disappearance of the finer vessels within its contour, and the narrowness of the retinal vessels that converge towards it, give unmistakable evidence that the nerve has passed into a condition of complete **atrophy**.

That in choked disc the subarachnoid liquid is forced into the arachnoid sheath of the optic nerve is inferred from the fact that, in cases of increased intracranial pressure, the sheath immediately behind the globe is distended by liquid into a kind of ampulla, in other words the condition is one of dropsy of the nerve-sheath. The pent-up liquid compresses the nerve behind the lamina cribrosa, impedes the efflux of blood through the central vein, and is supposed thereby to induce inflammation of the papilla. As however simple venous engorgement does not usually give rise to inflammation, it is not improbable that the simultaneous compression of the central artery diminishes the arterial blood-supply for a time, and so perhaps prepares the way for inflammation of the papilla. LEBER believes that the cerebral liquid forced into the arachnoid sheath has the property of directly exciting inflammation. It has been demonstrated by others (HUGUENIN) that progressive perineuritis of the trunk of the optic nerve may start from a tumour seated as far off as the temporal lobe, and that accordingly a creeping inflammation extending from the tumour to the meninges and to the optic nerves may be the cause of the papillitis. It is quite possible that a connexion of this nature between intracranial tumours and the optic papilla is more frequent than has hitherto been supposed.

Perineuritis of the optic trunk, such as occurs in orbital inflammation, basal meningitis, cerebral tumour (ZELLWEGER), and the like, is characterised by sero-cellular or fibrino-cellular exudation in the sheath of the optic nerve. The endothelial cells covering the delicate trabeculae that traverse the arachnoid space are multiplied, and in some instances the space is entirely filled up with an inflammatory exudation containing a large proportion of cells.

Interstitial neuritis is indicated by the cellular infiltration of the fibrous framework of the optic nerve: it often accompanies perineuritis, and leads to hyperplasia of the fibrous tissue and atrophy of the nerve-bundles. Lastly the inflammation, either independently of or accompanied by the two foregoing processes, may attack the nerve-bundles themselves, and terminate in grey degeneration of the optic nerve, the nerve-fibres wasting, their medullary sheaths breaking down, and the whole structure becoming pervaded by multitudes of fat-granule cells.

Syphilitic optic neuritis often follows upon specific retinitis (neuro-retinitis), but it may occur independently, either in the form of simple inflammation or of gummatous infiltration. The entire nerve or the optic chiasma alone may be permeated by gummatous growths.

Tuberculosis of the optic nerve has hitherto been rarely observed. It is however probable that miliary tuberculosis of the optic sheath, first observed by MICHEL and afterwards by DEUTSCHMANN, is commoner than is generally suspected. The experimental investigations of DEUTSCHMANN show that in rabbits at least tuberculous infection of the cranial cavity is rapidly followed by the appearance of miliary tubercles in the optic sheath behind the eyeball, though at first there are no signs of inflammation in the central portion of the optic trunk. Chronic tuberculosis of the nerve itself is rare, though occasionally a considerable segment of the trunk is found to be pervaded by tuberculous granulations, and so brought into a condition of atrophy.

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CHAPTER CXV

GLAUCOMA

371. **Glaucoma** is the name given to an affection of the eye in which the intra-ocular pressure is abnormally increased. In some cases the eye becomes as hard as stone. While in adults the globe remains practically unchanged in shape and size, in children it increases in circumference and may pass into the condition known as hydrophthalmos.

Glaucoma may supervene in an eye that was previously healthy (primary glaucoma), or in an eye already in some way diseased (secondary glaucoma). It may be acute or chronic, and with or without inflammatory symptoms. When the rise of the intra-ocular pressure takes place very slowly, without any reddening of the eye, and with occasional intermissions, the condition is termed **simple glaucoma**. In this case the only morbid changes observed are the excavation and atrophy of the papilla described below. But when the glaucoma runs an acute course, the exterior of the eye also exhibits marked changes: the ocular conjunctiva is intensely injected, and it may be even chemotic; the cornea is slightly turbid and smoky-looking, while its surface loses its brilliant lustre, and becomes dull and uneven; the pupil is usually somewhat dilated, and the faint greyish-green shimmer that shows through it, which is apparent also in the eyes of elderly people with large pupils, suggested the term glaucoma for the affection. Cases accompanied by the above-mentioned implication of the conjunctiva, together with distinct turbidity of the cornea and it may be of the vitreous also, in other words by the symptoms of ophthalmitis, are distinguished as cases of **inflammatory glaucoma**. In both forms, even when the rise of pressure is somewhat acute and severe, there may be no obvious pathological change other than hardness of the eye-ball, dulness of the cornea, and slight circumcorneal hyperaemia. In **haemorrhagic glaucoma**, with the symptoms already described are associated haemorrhages into the retina, the vitreous, the anterior chamber, and elsewhere.

The anatomical changes that the eye undergoes in glaucoma may be divided into primary and secondary. The former appear to be essentially of a chronic inflammatory character, while the latter are rather atrophic and degenerative in their nature.

According to some observers (VON ARLT, FUCHS, BIRNBACHER, Czermak, and others), chronic inflammation of the choroid, and particularly of its anterior portions, is the lesion that originates the accumulation of liquid and the consequent rise of pressure within the eye. According to others (Knies, Weber, Sargent, etc.) the principal factor in the process is a circumscribed inflammatory infiltration of the parts about the canal of Schlemm, which leads to the formation of new cellular tissue in Fontana's space (the most peripheral part of the anterior chamber) and upon the anterior surface of the iris. The result is that Fontana's space is filled up and the peripheral part of the iris becomes adherent to Descemet's membrane at the periphery of the cornea. As the principal outlet for the intra-ocular liquid flowing from the anterior of the eye is through Fontana's space, when this is obliterated stagnation and engorgement ensue, and the glaucomatous over-distension is induced. Other researches again have demonstrated the presence of well-marked changes in and about the vessels, and these have been regarded by several observers as the starting-point of the process, in particular by Klebs, who attributes much importance to the occlusion of the choroidal veins by thrombi composed

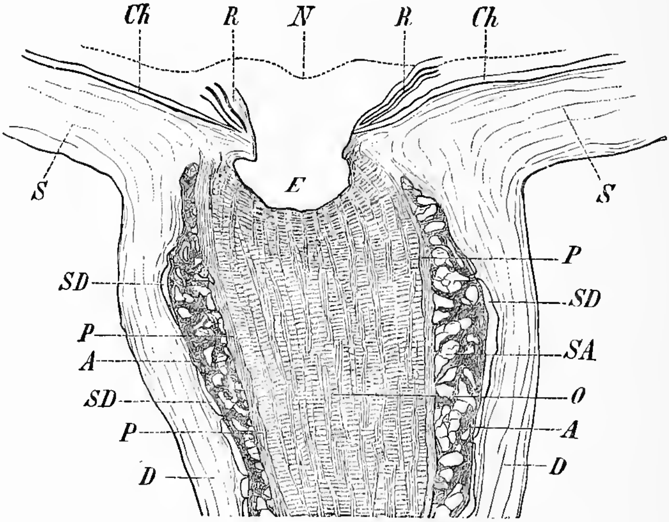

Fig. 562. Absolute glaucoma.

(*Longitudinal section through the optic nerve at its entrance into the eye; atrophy of the nerve-trunk:* × 12)

E	excavation of the papilla	*D*	dural sheath
N	line corresponding to the level of the normal papilla	*SA*	subarachnoid space
O	trunk of the optic nerve	*SD*	subdural space
P	its pial sheath	*S*	sclerotic
A	arachnoid sheath with thickened trabeculae	*Ch*	choroid
		R	retina

of blood-plates, which are gradually converted into hyaline thrombi. BIRNBACHER and CZERMAK detected signs of endothelial proliferation in the venae vorticosae, which in places almost completely filled up the vessels and so impeded the efflux of venous blood: the rise of intra-ocular pressure was then a natural consequence. But it is not yet sufficiently clear that any of these morbid changes in the glaucomatous eye are really primary, and not secondary to some undiscovered lesion.

Of secondary pathological changes, glaucomatous **excavation of the papilla** is that which must first be mentioned. As the lamina cribrosa is the weakest spot in the sclerotic, it is naturally the first to yield to the increased intra-ocular pressure, and is gradually forced backwards. At the same time the pressure upon the nerve-fibres of the papilla causes them to atrophy, and the depth of the excavation is thus still further increased. The depression at the site of the papilla (Fig. 562 *E*) varies from 0·6 to 1·5 millimetres in depth, and from 0·7 to 1·25 millimetres in width. The lamina cribrosa is occasionally forced back beyond the level of the outer surface of the sclerotic.

In the earlier stages the floor of the glaucomatous excavation is often covered with a layer of newly-formed tissue composed of small cells. This subsequently disappears, together with the subjacent nerve-fibres, so that the floor of the excavation is formed by the denuded lamina cribrosa (*E*): in the end the nerve-fibres and vessels along the sides of the excavation also disappear, and the condition known as **absolute glaucoma** is reached. When this condition has lasted for some time the trunk of the optic nerve also becomes atrophic, its diameter being considerably diminished, and the subarachnoid and subdural spaces of its sheath dilating proportionally (*SA* and *SD*).

In the retina the layers of nerve-fibres and of ganglion-cells are the first to atrophy, while the walls of the vessels become thickened, sclerotic, and varicose. DEUTSCHMANN found that in haemorrhagic glaucoma the retinal vessels were in some places plugged with red blood-corpuscles, and in others obstructed by small fibrinous coagula. He also observed well-marked hyaline thickening of the coats of the vessels, and their channels already contracted from this cause were often completely occluded by strands of slender fibrils.

The vitreous is in some cases unchanged, though its posterior segment is often detached from the retina. It occasionally encloses abnormal cells of irregular form, together with blood-corpuscles and pigment-cells.

The surface of the cornea in glaucoma, as in keratitis, becomes dull and slightly uneven, as if sprinkled with very minute drops of water. This appearance however is not due to inflammation but depends simply on the increased tension, and is therefore apt rapidly to appear and disappear. According to FUCHS, it is brought about by the deposition of small drops of liquid between the epithelial cells, particularly those of the deepest layer: the drops are often arranged in moniliform rows. Between the epithelium and Bowman's membrane a newly-formed structureless or fibrous-looking membrane is also frequently observed. In Bowman's membrane itself the canals giving passage to the nerves of the epithelium are dilated. Fuchs also detected shallow cleft-like lacunae in the cornea, lying parallel to its surface and increasing in size towards the anterior, which tended to force asunder the corneal laminae. According to this observer, all these appearances are due simply to oedema of the cornea, produced by the glaucomatous tension.

In **secondary glaucoma** dependent on other morbid processes, such as occlusion of the pupil, ectatic cicatrices of the cornea, sarcoma of the choroid, injury or dislocation of the lens, etc., the characteristic glaucomatous changes are superadded to those produced by the primary disease. Knies found, in cases of sarcoma of the choroid and of dislocation of the lens into the vitreous, that the space of Fontana was effaced; and Fuchs describes the same change in a case of sarcoma of the choroid.

The view that the obliteration of Fontana's space is the primary and essential factor in the production of glaucoma is not shared by all observers. Many regard it as merely a secondary effect arising from the pressure on the iris. A. Weber and Knies regard the occlusion of this space as a necessary and sufficient cause, but explain its production by supposing that the ciliary processes first become swollen, and so push forward the periphery of the iris, and that the latter then becomes adherent to the periphery of the cornea. Fuchs, Stölting, and others have shown by anatomical investigation that the ciliary body and its processes are actually enlarged. Priestley Smith holds that enlargement of the lens, such as he has demonstrated in the eyes of aged persons, is the primary cause of glaucoma. The enlarged lens obstructs the outflow of liquid from the vitreous towards the anterior chamber; the lens together with the zonula of Zinn and the ciliary processes are pushed forward, and the iris is pressed against the cornea; the effacement of Fontana's space is thus brought about, and glaucoma is induced. This last hypothesis fails however to explain the cases in which the lens is absent, owing for example to its dislocation into the vitreous. Further investigations are required to decide which of the above accounts of the pathogenesis of glaucoma is the true one, and whether the increased pressure is in all cases due to the same morbid condition.

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CHAPTER CXVI

TUMOURS AND PARASITES OF THE EYE

372. Of the **primary epithelial tumours** of the eye and its appendages, **squamous epithelioma** is the commonest. It usually starts at the junction of the conjunctiva and the cornea; but it may arise in the ocular conjunctiva (HORNER) or in that of the lids, and spread thence to the globe. So long as the carcinomatous growths are small they resemble pustules of eczema (VON GRAEFE); as they increase in size they assume the form of firm uneven tumours, which increase in width and in depth, and invade the sclerotic as well as the corneal margin. They are usually unpigmented, though melanotic carcinomata of this region have been repeatedly observed and described (HIS, LANGHANS, MANZ).

Carcinoma of the **lacrimal glands** (HORNER) is rare. It forms nodose tumours that force the eye to one side. By the development of hyaline spherules in the substance of the epithelial ingrowths the neoplasm described as *cylindroma carcinomatodes* is produced. **Adenoma** of the lacrimal glands has repeatedly been observed.

Carcinoma rarely invades the eye as a secondary growth: when an ocular metastasis does occur it is usually in the choroid, and as a rule the primary carcinoma is in the mamma.

Of the **connective-tissue tumours**, sarcoma and glioma are by far the most important. Myxoma, fibroma, lipoma, chondroma, osteoma, and the rest, are in comparison of much less account. Angioma and lymphangioma are also rare.

Sarcoma originates as a rule in the uveal portion of the iris, and is generally pigmented like the tissue from which it springs. Of the uveal sarcomata the majority arise in the choroid (85 per cent. according to FUCHS), and principally in its posterior segment; those of the ciliary body are less frequent (9 per cent.), and those of the iris comparatively rare. The proportion of pigmented forms to non-pigmented is given as 220 to 30. Nearly one-half of the recorded uveal sarcomata have been of the spindle-celled type; others included spindle-celled, stellate-celled, and round-celled tissue. The non-pigmented tumours are mostly of the round-celled type.

Sarcoma of the choroid starts in the pigment-bearing layer of large vessels. The chorio-capillaris and the lamina fusca are not invaded until afterwards. According to FUCHS, the neoplastic growth originates partly from the cells of the vascular adventitia and the endothelial intima, and partly from the pigmented cells of the stroma. The former produce the non-pigmented, the latter the pigmented tumour-cells, although pigment sometimes occurs in the derivatives of the former cells also. The growths usually form rounded nodes, which as they grow spread either outwards or inwards, or in both directions simultaneously. When the growth expands towards the interior, it first penetrates the lamina vitrea, the pigmentary epithelium remaining passive, and being pushed aside without taking any part in the elaboration of the tumour. The neoplasm then spreads into the sub-retinal space, or piercing the retina presses forward into the vitreous, producing large cells in great abundance, while the cells of the vitreous itself are speedily converted into sarcoma-cells. In a few cases the growth extends within the choroid in the form of small disseminated nodules.

When the sarcoma grows towards the exterior, it follows the course of the arteries, veins, and nerves that normally traverse the sclerotic. In the anterior segment of the eye the invasion proceeds mainly along the vorticose veins and the anterior ciliary nerves; at the fundus it proceeds by way of the numerous *arteriae posteriores breves.* Occasionally it reaches the exterior of the eye by following the optic nerve, either traversing the papilla and the lamina cribrosa, or extending directly backward from the margin of the choroid past the papilla into the arachnoid sheath of the nerve. The extra-ocular portion of the tumour always enlarges more rapidly than the part that is subjected to the intra-ocular pressure within the globe.

Sarcoma of the choroid invariably gives rise to metastases sooner or later, but usually at an early stage; these never appear in the neighbouring lymph-glands, but as a rule in the liver; secondary growths in the liver are indeed more frequent than in all the other organs taken together. The metastatic tumours are often less pigmented than the primary growth, and sometimes contain no pigment at all.

The morbid changes within the eye produced by choroidal sarcoma are, first, those characteristic of glaucoma, and secondly, inflammatory changes in the form of irido-cyclitis and choroiditis. In the latter case all the portion of the choroid that is not occupied by the new-growth is thickly infiltrated with leucocytes, the nuclei of the vessel-walls are proliferous, and so on. Not infrequently the surface of the choroid is overlaid with exudations, which later on become organised. In the fibrous tissue so formed ossification at times ensues. Detachment of the retina and cataract are occasional concomitants.

Sarcoma of the lacrimal glands, of the conjunctiva, or of the sclerotic is uncommon, though examples are now and then met with, some of them attaining a considerable size (Dyer, Nettleship). Conjunctival and sclerotic sarcomata usually start from the margin of the cornea and are in general pigmented.

In the trunk of the optic nerve both spindle-celled and round-celled sarcomata occur in the form of cylindrical, fusiform, or nodose enlargements. Lastly, tumours of this kind sometimes arise in the extra-ocular structures within the orbit or in its periosteum. Here also sarcomatous cylindroma, plexiform sarcoma, myxosarcoma, fibrosarcoma, and osteoma have from time to time been observed. They all assume the form of nodose tumours, which cause more or less marked protrusion of the eye-ball.

Perls recorded a case of true neuroma of the optic nerve, and Horner one of papillomatous fibroma of the conjunctiva.

Glioma appears chiefly in the retina, rarely in the trunk of the optic nerve, and is sometimes bilateral. While choroidal sarcoma is not met with before the second year, and very rarely before the tenth, retinal glioma occurs only in childhood (up to about the twelfth year), never in later life: it is indeed sometimes congenital, and its inception probably in most cases dates from the intra-uterine period.

The minute structure of retinal glioma corresponds in the main with that of cerebral glioma. The tumour is made up of closely-packed uninuclear cells embedded in a scanty intercellular stroma of finely-granular or fibrillar texture, which is traversed by numerous wide thin-walled vessels. In hardened preparations the glioma-cells look rounded, their nuclei being enclosed within a very small amount of protoplasm. Thus the bulk of the tumour seems to consist of little grains like those in the nuclear layers of the retina. Many authorities indeed maintain that the growth consists chiefly of lymphoid cells. On teasing fresh specimens Leber found that the cell-protoplasm was prolonged into numerous delicate interlacing fibrils, similar to those of Deiters' cells, and in striking analogy to the structure characteristic of glioma of the brain. Vetsch also found that this kind of cell was very often present, and the more frequently as the preparation was fresher. It would thus appear that retinal glioma is closely related to that of the brain, from which it however differs in one important particular, namely that it gives rise to metastases in various parts of the body, and tends to invade the contiguous tissues. It fills the entire globe, extends into the sclerotic, the eye-lids, and the soft parts and bones of the face, and may also spread along the optic nerve to the brain, following rather the course of the nerve-fibres than of the nerve-sheaths.

Animal parasites are rarely observed in the eye. In certain districts *Cysticercus cellulosae* is met with, usually occupying the interior of the eye-ball or very exceptionally the orbit. Intra-ocular *Cysticercus* is commonest beneath the retina and in the vitreous: it is in rare cases seen in the anterior chamber, where it is either freely movable or else attached to the iris or to Descemet's membrane. The parasite appears in this situation as a small transparent pale-yellowish vesicle, which moves about and from time to time protrudes its head and filiform neck. Even a subretinal *Cysticercus* shows movement of its walls and changes its position, though its head remains withdrawn into the vesicle. As seen with the ophthalmoscope, the vesicle appears as a white sharply-defined body often showing at some point a clear spot, which corresponds to the position of its head. When the vesicle attains a certain size, the overlying retina becomes opaque, and this is followed by more or less extensive detachment of the membrane, turbidity of the vitreous, and encapsulation of the vesicle in granulation-tissue containing giant-cells; this tissue is by and by converted into a dense fibrous cicatrix, and in the course of years may become partially calcified. The degenerate retina and choroid are also liable to become thickened by fibrous hyperplasia, and so blended with the encapsuling cicatrix. The parasite sometimes however forces its way through the retina into the vitreous, and in this situation may be seen as a freely-movable bluish vesicle, the head being sometimes drawn in and sometimes protruded. Such *Cysticerci* also soon produce turbidity of the vitreous, become encapsuled in fibrous tissue, and usually bring about detachment of the retina. Intra-ocular *Cysticercus* if left to itself leads finally to atrophy and destruction of the eye from plastic or suppurative irido-cyclitis.

The other ocular entozoa are *Filaria* and *Echinococcus*. The latter is occasionally met with in the orbit; the former has been observed in the anterior chamber and in the vitreous.

References on Epithelial Tumours of the Eye.

ALT: *Trans. Canada med. association* I Montreal 1877
BERLIN: *Graefe and Saemisch's Handbuch* VI Leipzig 1875
VON GRAEFE: *A. f. Ophth.* VII 1861
HORNER: *Klin. Monatsbl.* 1871
KNAPP: *Intra-ocular tumors* (trans. by COLE) New York 1869

References on Histioid Tumours of the Eye.

BIZZOZERO: *Moleschott's Untersuchungen* XI Frankfort 1870
DYER: Sarcoma of the ocular conjunctiva *Trans. Amer. Ophth. Soc.* 1879
FUCHS: *Das Sarkom des Uvealtractus* Vienna 1882
DA GAMA PINTO: *Untersuchungen über intraoculare Tumoren* Wiesbaden 1886
VON HIPPEL: Gliomatous metastases of the parotid *Bericht ophthalm. Univ.-Klinik* Giessen 1881
LEBER: Glioma *Graefe and Saemisch's Handbuch* V Leipzig 1875
SACHS, WOLF: Lymphangioma *Ziegler's Beitrage* V 1890
VETSCH: *A. f. Augenheilk.* XI 1881
VIRCHOW: *Die krankhaften Geschwülste* II Berlin 1863–7

References on Parasites of the Eye.

BERLIN: *Graefe and Saemisch's Handbuch* VI Leipzig **1875**
DOLINA: Intra-ocular cysticercus *Ziegler's Beiträge* V **1890**
HAAB: *Correspbl. f. Schweizer Aerzte* **1881**
TURNBULL: Filaria *Med. and Surg. Reporter* XXIX Philadelphia **1878**
VON WECKER: *Graefe and Saemisch's Handbuch* IV Leipzig **1875**

SECTION XV

THE EAR

(*by Professor* WAGENHÄUSER *of Tübingen*)

CHAPTER CXVII

MALFORMATIONS OF THE EAR

373. **Congenital malformations** of the ear may involve the entire organ or only particular parts of it: those of the external or middle ear are generally unilateral, and are often associated with other congenital defects, such as cleft palate, branchial fistulae of the neck, or facial hemiatrophy. They are in general referable to imperfect closure of the first branchial cleft in the foetus, from the margin of which the outer ear is developed.

The helix, antihelix, or lobe may be wanting, or the entire pinna may be stunted and deformed (microtism), or in rare cases absent altogether. Again the whole or part of the pinna is sometimes abnormally large, or surrounded by integumentary or cartilaginous supernumerary auricles (polyotism), or duplicated. It is moreover liable to be misplaced, so that it appears to grow from the cheek, the neck, or even the shoulder.

Persistent traces of the first branchial cleft, in the form of small scar-like pits and fistulous tracks or sinuses that secrete a creamy liquid, are sometimes present near the ear (*fistula auris congenita*), but they do not communicate with the external meatus or the tympanic cavity (URBANTSCHITSCH).

Complete absence of the external auditory meatus (*atresia congenita*) occurs as a rule only in connexion with absence or extreme undergrowth of the pinna, and is generally associated with malformation of the tympanic membrane and tympanum. The place of the absent meatus is occupied by a compact mass of bone, with a shallow depression indicating the position of its external opening. In other cases there is a cartilaginous canal, which however in its deeper portions is either closed by bone or membrane or continued as a filiform channel. Uniform or hourglass constriction, fibrous bridges connecting the sides of the meatus, and duplication or abnormally-wide calibre of its channel, are among the other anomalies that have been recorded.

The tympanic membrane also is subject to various anomalies of form, size, and position. Congenital absence of it is always associated with absence of the external meatus and tympanic cavity. Arrest of development occasionally gives rise in both ears to a deficiency in the membrane at its junction with the *membrana flaccida* of Shrapnell (*foramen Rivini*).

The tympanic cavity with its ossicles is in some cases rudimentary or entirely absent: it may be contracted to a mere cleft, its fenestrae may be absent or defective, or its cavity may be abnormally wide or even duplicated. Ankylosis of the incus with the stapes, or of all three ossicles into a single *columella*, is of rare occurrence. Of the three bones the stapes is the one that is most apt to be misshapen.

Malformations of the eustachian tubes are somewhat uncommon; entire absence of them has been noted in association with absence of the external ear and with rudimentary conditions of the tympanum and labyrinth. Congenital occlusion and stenosis of one tube are likewise infrequent, though such anomalies as angular flexure of the channel and malposition of the pharyngeal orifice are somewhat more common.

The mastoid process may be defective or absent: while variations in its external size or shape, such as unciform curvature, and abnormal size, form, and distribution of the mastoid cells, are by no means rarely observed.

Among malformations of the internal ear, whether associated with defects of the external and middle ear or not, the following are the more important that have been described—Absence of the labyrinth, absence or rudimentary condition of some or all of the semicircular canals, absence of the cochlea and of its modiolus or lamina spiralis, and duplication or dilatation of the aqueducts. When the labyrinth is absent the auditory nerve is either wanting or ends in a bulbous enlargement within the petrous bone.

References on Malformations of the Ear.

BURNETT: *The ear* London and Philadelphia **1884**

GRUBER: *Lehrb. d. Ohrenheilk.* Vienna **1870**

ISRAEL: Congenital fissures of the lobe *V. A.* 119 **1890**

JOËL: Congenital atresia *Z. f. Ohrenheilk.* and *A. of Otol.* XVIII **1889** (with references)

LESSER: Rare anomalies *D. Z. f. Chir.* II **1873**

PAGET: Branchial fistulae *Med.-chir. Trans.* LXI London **1878**

POLITZER: *Lehrb. d. Ohrenheilk.* Stuttgart **1878** (trans. by CASSELLS) London **1883**

SCHAEFFER: Foetal development of the ear *A. f. Anthrop.* XXI **1892**

SCHWARTZE: Pathological anatomy of the ear *Klebs' Handb. d. path. Anat.* part 6 II Berlin **1878** (trans. by ORNE GREEN) Boston **1878**

THOMSON, ALLEN: Malformations of the external ear *Monthly J. of med. science* I Edinburgh **1847**

VON TRÖLTSCH: *Lehrb. d. Ohrenheilk.* Würzburg **1868** (trans. by HINTON) London **1874**; 7th edition Leipzig **1881**

URBANTSCHITCH: *Lehrb. d. Ohrenheilk.* Vienna **1890**

VIRCHOW: *V. A.* 30 **1864** and 32 **1865**

CHAPTER CXVIII

THE EXTERNAL EAR

374. **Hyperaemia** of the integument of the outer ear often occurs in paretic and paralytic conditions of the sympathetic and vaso-motor nerves of the cervical plexus. Mechanical irritation readily causes hyperaemia of the external meatus and tympanic membrane.

Haemorrhage into the tissue of the pinna and the parts about it gives rise to **othaematoma**, a bluish-red fluctuant swelling overlying the concave aspect of the ear, due to effusion of blood between the cartilage and the perichondrium. Two varieties are distinguished, one traumatic, in which the cartilage is generally injured or fractured, the other spontaneous, and occasionally bilateral and symmetrical. The latter affection is nearly always associated with mental disorder, though it sometimes occurs in sane persons also. It is probable that degenerative changes in the cartilage, leading to softening and vascular proliferation, predispose to the condition. BROWN-SÉQUARD regarded spontaneous othaematoma as indicating a lesion at the base of the brain, experiments on the lower animals having shown that section of the restiform bodies is followed by haemorrhage beneath the skin of the ear.

Subcutaneous extravasations in the external meatus, assuming the form of small ecchymoses or of larger haemorrhagic bullae, result from mechanical injury or from intense inflammation of the meatus itself or of the adjoining cavities of the tympanum or mastoid process.

Cysts of the pinna following othaematoma have been described by HESSLER (*A. f. Ohrenheilk.* XXIII **1886**), and very probably the cystic changes referred to by HARTMANN are connected with the same condition (*Z. f. Ohrenheilk.* XV **1886** and XVII **1888**).

References on Othaematoma.

BIRD: *Journ. d. Chir. u. Augenheilk.* XIX Berlin **1833**
BROWN-SÉQUARD: *Bullet. Acad. de méd.* XXXIV
FRÄNKEL: *V. A.* 99 **1885**
GUDDEN: *Allgem. Z. f. Psych.* XVII **1860** XIX **1862**
HAASE, G.: *Z. f. rationelle Med.* 3rd series XXIV **1865** (with references up to 1864)
HAUPT: *Inaug. Diss.* Würzburg **1867**

HUN: Twenty-four cases *Amer. J. of Insanity* XXVII **1870**
MEYER, L.: *V. A.* 33 **1865**
SCHWARTZE: Spontaneous variety in sane persons *A. f. Ohrenheilk.* II and III **1867**
VIRCHOW: *Die krankhaften Geschwülste* I Berlin **1863**

375. **Inflammation** of the skin of the ear is either a primary affection or arises by extension from some inflammation in the adjacent parts, such as one of the acute eruptions, erysipelas, erythema, or eczema, or less frequently herpes, lupus, syphilitic pemphigus, and ichthyosis. Gangrene of the integument has been known to accompany typhus fever and erysipelas, and at times it occurs spontaneously in infants.

Furunculosis of the external meatus is of frequent occurrence: it is a circumscribed inflammation of the hair-follicles due to infection by the pyogenic *Staphylococci*, and is sometimes epidemic.

Diffuse inflammation affecting a large part or the whole of the lining of the external meatus, and manifested by redness, swelling, and serous or purulent exudation, may be set up by chemical and thermal irritants, or by traumatic injury: it also occurs in connexion with acute and chronic cutaneous eruptions, erysipelas, and purulent discharges from the middle ear.

Croupous inflammation of the meatus occurs in rare instances as an idiopathic affection. It affects chiefly the outer surface of the tympanic membrane and the bony portion of the canal, spreading thence outwards. The croupous false-membrane forms a stiff tenacious cast of the canal, and when detached leaves behind a bleeding and excoriated surface. The proneness of the meatus to croupous inflammation is regarded by BEZOLD as depending on the thinness of its epidermis and the liability of its lining membrane to intense hyperaemia.

Diphtheritic inflammation is usually an accompaniment of the like affection in the mucous membrane of the throat and middle ear. It occasionally occurs as a primary or independent affection, when abrasions of the meatus afford an entrance to the infection of diphtheria.

Syphilitic inflammation gives rise to broad reddened papules, whose surface is at first dry but afterwards 'weeps,' seated chiefly about the external opening of the meatus. If suppuration of the middle ear is in progress at the same time the papules are apt to break down, and form spreading ulcers overlaid with a gristly-looking film.

Both the diphtheritic and the syphilitic affections result at times in cicatricial contraction or even occlusion of the meatus.

Desquamative inflammation is associated with the exfoliation of successive strata of the epidermis and epithelium in the deeper portions of the meatus and in the tympanic membrane, and gives rise to firm adherent deposits consisting of white glistening concentrically-stratified lamellae.

Mycotic inflammation, which chiefly affects the inner third of the canal and the tympanic membrane, causes the surface to be overlaid by macerated masses of epidermal scales that are pervaded and overgrown by mycelial filaments (Art. 377), the corium beneath being reddened and denuded.

Phlegmonous inflammation of the pinna and external canal is due to the septic infection of traumatic lesions.

As the deeper layers of the lining membrane of the inner portions of the canal subserve the function of a periosteum for the underlying bone, all the deep-seated inflammations of these parts may be regarded as forms of periostitis. Apart from this, periostitis of the meatus is usually an accompaniment of acute inflammation of the tympanic cavity and mastoid process.

Perichondritis of the pinna and the external meatus is of rare occurrence: in the former situation it produces a red fluctuant swelling, resembling an othaematoma, occupying the concavity of the pinna and stopping abruptly just above the lobe.

Caries of the walls of the meatus sometimes follows one or other of the above-mentioned inflammations; but it more frequently results from chronic suppuration of the tympanic cavity and mastoid process. The pus as it burrows behind or above the lining membrane causes the latter to bulge into the lumen of the canal in loose sacculations: and when these give way they leave fistulous tracks proceeding from the meatus into the bone, and in severe cases communicating with the middle ear. Their usual position is in the posterior superior part of the wall near the tympanic membrane, whence they lead into the mastoid antrum, or in the roof of the meatus just above the tympanic membrane.

Necrosis of the bony structures is not uncommonly induced in a similar way, and occasionally results in the exfoliation of considerable fragments of bone. Thus in children the entire tympanic ring with the adjacent parts of the mastoid and squamous bones is sometimes separated and extruded *en masse*. Fistulous tracks in the meatus may also be produced by the bursting of parotid abscesses, or of abscesses due to periostitis of the mastoid, through the *incisurae Santorini* in the cartilage or through the junction of the cartilaginous and osseous portions of the meatus.

Hyperostosis of the external meatus, giving rise to considerable constriction of its channel, is of frequent occurrence in cases of chronic otitis media, purulent or non-purulent.

It is worth bearing in mind, inasmuch as it is sometimes set down as evidence of caries, that in children there is normally a lacuna, filled in with fibrous tissue, in the anterior wall of the bony canal. The lacuna usually persists until the fourth year or sometimes even longer, and its existence renders it possible for inflammation of the external canal to extend into the parotid gland and the temporo-maxillary joint (VON TRÖLTSCH: *Lehrbuch* Leipzig **1881**; and BÜRCKNER: *A. f. Ohrenheilk.* XIII and XIV **1878**).

References on Furunculosis.

Loewenberg: *Le furoncle de l'oreille et la furonculose* Paris **1881**, and *D. med. Woch.* 28 **1888**
Schimmelbusch: Aetiology of furunculosis *A. f. Ohrenheilk.* xxvii **1889**
Vermyne: Epidemics *Amer. J. of Otol.* iv **1882**

References on Croupous and Diphtheritic Inflammation.

Bezold: *V. A.* 70 **1877**
Guranowsky: *Monatsschr. f. Ohrenheilk.* xxii **1888**
Kraussold: *Cent. f. Chir.* **1877**
Moos: *A. f. Augen- und Ohren-heilk.* Wiesbaden **1871**, and *A. of Ophth. and Otol.* New York **1871**
Steinhoff: *Inaug. Diss.* Munich **1886**
Wreden: *Monatsschr. f. Ohrenheilk.* ii **1868**

References on Syphilitic Inflammation.

Buck: Thirty cases of syphilis of the ear *Amer. J. of Otol.* i **1879**
Dalby: *Lancet* i **1877**
Gruber: *Wien. med. Presse* xi **1870**
Knapp: *A. of Otol.* **1879**, **1880**, and *Z. f. Ohrenheilk.* ix **1880**
Schwartze: *A. f. Ohrenheilk.* iv **1869**
Stöhr: *A. f. Ohrenheilk.* v **1870**

376. Among **degenerative changes** affecting the external ear are included cleavage, softening, partial calcification, and ossification of its cartilage. In gout urates are deposited as *tophi* in the subcutaneous tissue.

The **tumours** met with in this situation include atheroma (sebaceous cyst), papilloma, fibroma, lipoma, angioma, enchondroma, sarcoma, and carcinoma.

Osteoma of the external ear is especially common, and assumes the form of pedunculated or sessile globular or conical tumours, sometimes spongy in structure, sometimes of ivory-like hardness. They generally spring from the posterior superior wall of the canal close to the tympanic membrane, and grow either singly or in groups of three or more.

The polypous histioid tumours of the meatus will be considered in connexion with aural polypi (Art. 382).

References on Tumours of the External Ear.

Blake: *Amer. J. of Otology* ii **1880**
Kikuzi: Keloid *Beiträge von Bruns* **1888**
Launay: Enchondroma *Gaz. des hôpitaux* **1861**
Moos: *Tagebl. d. Naturforscherversamml.* Freiburg **1883**
Schwartze: *Lehrbuch* (trans. by Orne Green) Boston **1878**
Toynbee: Atheromata *Med.-chir. Trans.* London xliv **1861** xlvii **1864**
von Tröltsch: *Lehrbuch* Leipzig **1881**
Weber, C. O.: *Die Exostosen und Enchondrome* Bonn **1856**
Welcher: *A. f. Ohrenheilk.* i **1864**

377. **Vegetable parasites** seem to have a kind of predilection for the external auditory canal. In addition to the common and long-recognised species of *Aspergillus* (*A. niger*, *A. flavus*, and *A. fumigatus*), and to the rarer *Ascophora elegans*, *Trichothecium roseum*, and *Eurotium repens*, the following have in recent years been described: *Aspergillus nidulans* (Eidam, Siebenmann), *Penicillium minimum* (Siebenmann), *Verticillium Graphii* (Bezold), *Mucor corymbifer* (Hückel), *Mucor septatus* (Bezold), and *Eurotium malignum* (Lindt).

The fungi, with their velvety pile of mycelial filaments, spread in a compact mass over the walls of the external canal. As they do not develope upon a healthy surface, their growth indicates the presence of some morbid condition of the lining membrane, and of certain abnormal secretions, such as superficial inflammation with serous effusion. They do not penetrate into the deeper-lying tissues, and though they no doubt cause irritation they should be regarded as saprophytes rather than as true parasites. For a description of the inflammatory changes induced by their presence see Art. 375.

The *Acarus folliculorum* is as yet the only **animal parasite** of the external meatus that has been observed in man.

Foreign bodies, such as peas, seeds, beads, small stones, and the like, often gain entrance into the auditory canal, and sometimes remain there without causing much disturbance. At times, however, when they swell up and exert pressure, they induce disorders of the circulation or cause reflex irritation involving the trigeminus and vagus nerves, and in this way give rise to epileptic or paralytic symptoms.

Disorders of secretion in the sebaceous and ceruminous glands of the cartilaginous and first part of the osseous canal are indicated either by deficiency of the cerumen and sebum, producing excessive dryness of the skin (as in trophic disorders associated with disease of the middle ear), or by abnormal abundance of these. The impacted masses that occupy the meatus under certain favouring conditions, such as stenosis of the canal and imperfect cleanliness, consist in some instances simply of inspissated secretion; in other cases they include epidermal scales, masses of fungi, and hairs. Concretions of calcium carbonate and phosphate (otoliths) are seldom found in the human meatus; they are more frequent in some of the lower animals.

In animals like the cow, sheep, dog, cat, and rabbit, the animal parasites known as *Dermanyssus avium*, *Dermatodectes*, *Symbiotes felis*, and *Gregarinida* are often met with (von Tröltsch: *Lehrbuch*); they all give rise to intense inflammation. In man the presence of purulent otorrhoea appears to render the external canal a favourite laying-place for flies: their larvae or maggots, and especially *Muscida sarcophaga* (Urbantschitsch: *Lehrbuch*) sometimes develope within it in large numbers. The accidental intrusion of fleas or bedbugs, and even of the dreaded earwig (*Forficula auricularis*), produces no results of any pathological importance.

References on Parasites of the External Ear.

BEZOLD: Fungi *Monatsschr. f. Ohrenheilk.* VII **1873**

BLAKE: Maggots *A. of Ophth. and Otol.* II **1872**; Fungi *Trans. Amer. Otol. Soc.* I **1871**

BURNETT: *The ear* London **1884**

CRAMER: Aspergillus *Vierteljahsschr. d. naturforsch. Gesellsch.* Zürich **1859–60**

EIDAM: *Cohn's Beiträge zur Biologie der Pflanzen* part 3 III Breslau **1881**

HAUG: Aspergillus nigrescens *Ziegler's Beiträge* XVI **1894**

HENLE: Acarus folliculorum *Müller's A. f. Anat. und Physiol.* **1842**

HINTON: *Questions of aural surgery* London **1874**

HÜCKEL: Mucor corymbifer *Ziegler's Beiträge* I Jena **1884**

LINDT: *A. f. exp. Path.* XXV **1889**

POLITZER: Aspergillus within the tympanum *Wien. med. Woch.* **1870**

SCHWARTZE: Aspergillus *A. f. Ohrenheilk.* II **1867**

SIEBENMANN: *Die Fadenpilze etc. und ihre Beziehung zur Otomykosis* Wiesbaden **1883**, and *Z. f. Ohrenheilk.* XIX **1890**

STEUDENER: Fungi *A. f. Ohrenheilk.* V **1870**

WREDEN: Aspergillus glaucus *A. f. Ohrenheilk.* III **1867**; *Die Myringomycosis* St Petersburg **1868**; *A. of Ophth. and Otol.* IV **1874**

CHAPTER CXIX

THE TYMPANIC MEMBRANE

378. The **tympanic membrane** is covered on its outer surface by a thin dermoid layer or cuticle continuous with that of the external meatus, and on its inner surface by the mucous membrane of the tympanic cavity. As its nutrition is maintained from both sources, it is usually involved by continuity when these cavities are diseased, primary and independent lesions of the membrane being on the whole infrequent.

Hyperaemia affects either the dermoid or the mucous layer singly, or both simultaneously. In slight hyperaemia a line of injected vessels is seen descending behind the handle of the malleus, while radial vessels converge from the periphery and anastomose with those of the malleus. When the hyperaemia is more intense the separate vessels of the dermoid layer are no longer apparent, the tympanic membrane being diffusely reddened.

Haemorrhage takes place either spontaneously or from traumatic injury, both in the outer and in the inner layer of the membrane: it appears in the form of punctiform and linear ecchymoses or of more copious extravasations (haematoma). In the mucous layer these are visible as bluish-red sharply-circumscribed elevations of the surface, and are met with chiefly in connexion with typhoid fever, small-pox, scurvy, and endocarditis. Ecchymoses of the dermoid layer gradually become displaced towards the periphery, usually backwards and upwards, and so on to the wall of the meatus.

Isolated and independent **inflammation** of the tympanic membrane (myringitis) is comparatively uncommon; it is usually associated with inflammation of the middle ear, or results from injury. In the acute form the drum-head looks intensely red and thickened, and the contour of the handle and short process of the malleus is no longer to be traced. Maceration and desquamation of the epidermis follow, and the corium is denuded and exposed. Under the microscope the dermoid layer appears infiltrated with round-cells, the middle layer (*substantia propria*) is swollen and relaxed, and the mucosa is turgid from congestion and cellular infiltration. Circumscribed accumulations of pus-cells in the tissues of the cuticle often lead to the formation of small **interlamellar abscesses**, which appear as yellowish flattened elevations of the membrane. In chronic inflammation the membrane becomes thickened, its vessels are dilated and varicose, and its external surface is often covered with granulations and with small papillary or villous growths (*myringitis villosa*).

Cases of **tuberculous inflammation**, manifested by the formation of small yellowish translucent and somewhat prominent spots on the membrane, have been recorded in connexion with miliary tuberculosis and with tuberculous phthisis in adults.

Inflammation of the tympanic membrane often leaves behind it some permanent **opacity**, whereby the surface becomes studded with irregular whitish spots, due to the formation of new connective tissue, to thickening of the epidermis, and to fatty infiltration.

Calcification of the membrane, assuming the form of yellowish-white sharply-circumscribed spots, is also a very common result of inflammation. The deposits sometimes extend over the entire membrane, and are usually seated in the middle layer, the calcareous particles being clustered in the tubular sheaths surrounding its fibrils: sometimes the deposits are met with in all three layers, but rarely in the outer or inner layer alone. **Ossification** of the tissue of the membrane is seldom observed.

Diffuse thickening of the tympanic membrane, occasionally to the extent of five times its normal depth, is a common result of cellular infiltration of the submucosa of the inner layer.

Atrophy of the membrane is an occasional consequence of long-continued stretching (as from persistent retraction due to occlusion of the eustachian tube), or of the pressure of morbid deposits against it: the middle layer or substantia propria is that which tends to waste and disappear. Local and partial atrophies are distinguishable from cicatricial patches only by their less sharply-defined outlines. When it is atrophied throughout the membrane collapses inwards and is closely applied to the deeper tympanic structures, which can be seen through it; in some instances it bulges outwards like a bladder when the air behind it is compressed.

Anomalies of **curvature**, with the convexity outwards, are occasionally due to changes such as swelling, thickening, or interlamellar abscess, affecting the membrane itself; but they are more frequently brought about by the pressure of exudations accumulating in the tympanic cavity. Concave retraction and flattening of the normal infundibular form of the membrane result from thickening of the mucous layer on the one hand, or from extreme atrophy on the other: in the latter condition it is apt to sink deeply into the tympanic cavity. Marked prominence of the short process, and foreshortening of the handle of the malleus as viewed from without, are indicative of excessive retraction, resulting it may be from changes in the tympanic cavity, long-continued occlusion of the eustachian tube, bands of adhesion, and shortening of the tensor-tympani tendon.

Cholesteatoma is a somewhat uncommon kind of tumour met with on the internal surface of the tympanic membrane: the growth is built up of a number of whitish scales arranged like the coats of an onion, and enclosed in a thin vascular membrane (Art. 382).

Polypous growths may arise either from the mucous or from the dermoid layer (Art. 382).

Circumscribed warty overgrowths of the tympanic epidermis, and pearly epithelial structures of about the size of a pin's head that are seated on the external layer, are sometimes met with; but they are of no great importance.

Perforation of the membrane, when it is not due to traumatic injury, or to atrophy of spontaneous origin or resulting from pressure, is dependent on inflammatory changes. Inflammation of the membrane itself may lead to perforation (as by the rupture of an interlamellar abscess); but much more frequently the perforative inflammation originates in the middle ear and thence extends to the membrane, which becomes infiltrated and under the influence of the primary irritant probably necrotic in parts. The pressure of the exudation collected within the tympanic cavity then causes it to give way, and the perforation is gradually enlarged by progressive disintegration of the tissue surrounding the rent.

The usual position of the perforation is in the zone between the handle of the malleus and the fibrous peripheral ring known as the *margo tympanicus*, though it may occur at any point. Usually there is but one perforation, though multiple ruptures have been observed in cases of typhoid fever, tuberculosis, and pyaemia. The size of the opening varies from the minutest puncture to practically the entire extent of the membrane. Repair may take place without any permanent change of a visible kind being left, as in the case of small and sudden perforations unaccompanied by any extensive destruction of the tissue of the middle layer. Larger perforations of some standing can be repaired only by a process of cicatrisation. On the other hand callous thickening or skinning over of the edges of the opening, and adhesion of the ruptured membrane to the wall of the tympanic cavity, are apt to prevent the closure of the perforation.

The cicatrix when fully formed consists of a thin stratum of fibrous tissue covered on both sides by large squamous epithelial cells, the substantia propria or middle layer not being reproduced. Owing to the absence of this, the strongest layer of the three, the scar appears as if depressed below the general level: it is sharply-outlined and of darker colour than the rest of the membrane. Cicatrices of any size tend to sink inwards and become adherent to the wall of the labyrinth. Calcareous deposits have more than once been observed in such cicatrices.

Laceration of the tympanic membrane is generally brought about by direct or indirect violence, such as the thrusting in of solid objects, extreme fluctuations of the atmospheric pressure, or concussion of the skull with or without fracture of the temporal bone. The laceration appears as a gaping wound of the membrane, or as a rounded clean or ragged perforation whose edges are suffused with blood.

Rumler has shown by experiments on rabbits that during the first three days after rupture the external epidermis, and then the epithelium of the mucous layer, alone take part in the reparative process. From the third day onwards the proliferation of the fibrous tissue is the most prominent feature, and it is this that effects the ultimate closure of the wound.

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Gottstein: Desquamative inflammation *Otol. Congress* (Milan 1880) Trieste **1882**
Gruber: *Monatsschr. f. Ohrenheilk.* **1871–1878**
Habermann: Tuberculosis *Prager Z. f. Heilk.* **1885**
Hinton: *Guy's Hosp. Reports* XII **1866**; Cholesteatoma *A. f. Ohrenheilk.* II **1867**
Jones, Macnaughton: *Atlas of diseases of the membrana tympani* London **1878**
Kessel: Myringitis villosa *A. f. Ohrenheilk.* V **1870**
Lucae: Aragonite-crystals in the thickened dermoid layer *V. A.* 36 **1866**
Marian: Abscess *A. f. Ohrenheilk.* XVII **1881**
Moos: *Klinik d. Ohrenkrankh.* Vienna **1866**; Traumatic haematoma *Z. f. Ohrenheilk.* VIII **1879** and *A. of Otol.* VIII New York **1879**
Nasiloff: Myringitis *Cent. f. med. Wiss.* **1867**
Politzer: *Beleuchtungsbilder des Trommelfelles* Vienna **1865** (trans. by Mathewson and Newton) New York **1869**, and *Lehrbuch* Stuttgart **1878**
Rumler: Regeneration of the tympanic membrane *A. f. Ohrenheilk.* XXX **1892**
Schwartze: *Pathological anatomy of the ear* (trans. by Orne Green) Boston **1878**
von Tröltsch: *Lehrb. d. Ohrenkrankh.* Leipzig **1881**; *V. A.* 17 **1859**
Urbantschitsch: *Lehrb. d. Ohrenheilk.* Vienna **1890**; Epithelial deposits *A. f. Ohrenheilk.* X **1875**
Wendt: Endothelial cholesteatoma *A. d. Heilk.* XII **1874**
Wette: Acute desquamative myringitis *Monatsschr. f. Ohrenheilk.* XVI **1882**
Wirtz: The relation of the foramen Rivini to purulent otitis *Inaug. Diss.* Bonn **1886**
Zaufal: Congenital bilateral perforation *Wien. med. Woch.* **1868**

CHAPTER CXX

THE MIDDLE EAR

379. **Hyperaemia** of the mucous membrane of the middle ear is often dependent upon affections of the pharynx, external auditory canal, labyrinth, and cranial cavity, owing to the intimate connexion between their blood-vessels. Cardiac disease, pulmonary affections, and tumours pressing upon the cervical vessels, are apt to give rise to diffuse venous engorgement of the membrane.

Haemorrhage, in the form of small ecchymoses or free effusion into the cavities of the middle ear, occurs both spontaneously and as the result of traumatic injury (violent concussion, fracture of the skull, etc.). Bleeding may also take place in association with severe inflammation of the middle ear, or with Bright's disease, diphtheria, leukaemia, and endocarditic embolism of the stylomastoid artery. Somewhat copious bleeding, with effusion of blood through the external meatus and eustachian tube, occasionally results (apart from fracture of the temporal bone) from spontaneous exfoliation of aural polypi and from caries of the tympanic wall with erosion of the carotid, jugular vein, and lateral or superior petrosal sinuses, such erosion often leading to fatal haemorrhage.

Owing to the continuity of its mucous membrane, **inflammation** (*otitis media*) generally extends to all parts of the middle ear, though in different degrees. Inflammation confined solely to the eustachian tube, the tympanic cavity, or the mastoid process is rare, the most characteristic, and in view of its relations the most important, affection of this region being inflammation of the tympanum itself.

Pathogenic microbes play an important part in the causation of **otitis media.** In the majority of cases the *Diplococcus pneumoniae* of FRÄNKEL and WEICHSELBAUM, and *Streptococcus pyogenes*, can be detected in the intra-tympanic exudation; next in frequency are *Staphylococcus pyogenes albus* and *aureus*, the *Pneumobacillus* of FRIEDLÄNDER, and *Bacillus pyocyaneus*. The pathogenic significance of *Staphylococcus cereus albus* and *Micrococcus tetragenus* is not yet clearly established. At times several species of bacteria are present together, or a second species proceeds to develope after the first has disappeared.

The channel by which microbes gain access into the tympanic cavity is in most instances the eustachian tube. Along this channel inflammation of the mucous membrane of the naso-pharynx is apt to extend, and infective matters are occasionally forced into it by blowing the nose, choking, or vomiting. Infection may also be conveyed by way of the blood in various infective diseases, such as measles, scarlet fever, diphtheria, small-pox, and influenza. Perforation of the tympanic membrane again sometimes opens a way through the external meatus whereby infection from without may reach the cavity.

Serous and mucous catarrh is usually associated with catarrhal affections of the nose and naso-pharynx. The mucous membrane on examination appears hyperaemic and swollen, and there is more or less abundant cellular infiltration of the submucosa, the inflammatory changes being either diffuse and extending over the entire membrane, or limited to certain parts of it. Serous liquid or tenacious stringy mucus, mingled with a small proportion of epithelial cells, mucus-corpuscles, and pus, fills the cavity and adheres to the ossicles, the tympanic membrane, and the fenestrae. In the chronic form the mucous membrane is thickened, tough, and greyish-white, and beset in places with small excrescences and villous outgrowths. The thickening is occasionally limited to the tympanic membrane, the ossicles, or the fenestrae; in other cases it extends over the entire mucous membrane.

Minute perforations of the tympanic membrane are now and then produced in cases of serous and mucous otitis media, but as a rule they rapidly reunite and heal up again.

The acute form of purulent catarrh is met with chiefly in the course of the acute exanthemata, such as measles, scarlet fever, and small-pox, and at times in association with diphtheria, typhoid fever, and tuberculosis. The anatomical changes it produces are similar to those observed in mucous catarrh, but they are more pronounced, and all the cavities of the middle ear appear to be filled with purulent exudations mingled with blood and mucus. Usually the inflamed and softened tympanic membrane is ruptured, and the pus thus escapes to the exterior; indeed it is only when the membrane has been much thickened and so toughened by previous chronic myringitis that it fails to rupture and let out the pus.

Extensive **ulceration** of the mucous membrane is comparatively rare; it is only indeed in connexion with septic or gangrenous inflammation that the mucosa is destroyed and the ossicles and bony walls of the tympanic cavity undergo carious erosion. Owing to the close proximity of the brain and the frequent presence of lacunae in the bony roof of the middle ear, as well as to the manifold vascular intercommunications between the tympanic and cranial cavities, purulent meningitis and phlebitis of the intracranial sinuses are liable to follow upon purulent otitis media, even though the bone itself is not attacked. The risk is of course increased when the drum-head is so thickened as to prevent the external evacuation of the pus.

Croupous and diphtheritic otitis are usually consecutive to the like affections of the naso-pharynx, the false-membranes sometimes extending along the eustachian tube as far as the mastoid process.

The occasional supervention of tuberculous otitis has been definitely proved to be possible by the detection of tubercle-bacilli in the purulent discharges and in the diseased mucous membrane of the tympanic cavity; the possibility had already been suggested by clinical observations.

Chronic purulent otitis media is characterised by many important anatomical features. The secretion, which is sometimes scanty, sometimes profuse, has a penetrating disagreeable odour, and is often coloured brown by admixture with blood. The tympanic membrane loses much of its substance by the softening and disintegration of the ruptured edges of the perforation. The lining membrane of the tympanum is often greatly thickened, the submucosa especially being densely infiltrated with round-cells, and its vessels dilated and multiplied, while the periosteal stratum is less affected. In some parts of the mucosa the ciliated epithelium is still intact, in others it is covered with epidermoid cells due to the ingrowth of the dermoid layer of the tympanic membrane round the edges of the perforation, in others again the surface is denuded of epithelium and overlaid with livid pus-secreting granulations. Local hyperplasia, with the formation of villous and fungating excrescences, gives the surface an uneven granulated appearance, and as the excrescences grow together they enclose small cyst-like cavities lined with epithelium. It is not uncommon also to find evidence of progressively-destructive changes issuing in ulceration of the mucous membrane, and owing to the intimate relation of the latter structure to the bone the ulceration readily deepens and induces caries and necrosis. The destruction is sometimes narrowly circumscribed and confined to special parts of the tympanum, but in other cases it involves the greater portion of the temporal bone.

Caries is most apt to attack the mastoid process: at times it affects only the partitions between the cells, and as these break down the interior of the bone is converted into a single large cavity filled with granulations and necrotic sequestra; but oftener the cortical layer of the bone is likewise diseased, and the pus breaks through to the exterior or interior. External evacuation generally takes place through the posterior superior wall of the bony meatus, or through the outer surface of the mastoid at its upper part or near the mastoid fissure, or on the inner aspect of its apex. Burrowing abscesses may thus be formed beneath the integument of the auditory canal or of the mastoid prominence.

Within the tympanic cavity caries usually attacks the upper surface about the tegmen tympani. When it involves the labyrinth-wall, the aqueductus Fallopii or the labyrinth itself may be broken into, and facial paralysis or extension of the inflammation within the skull may thereupon take place. Often too the bony partition that divides the socket of the head of the malleus from the external meatus is destroyed by ulceration; but the floor of the tympanum and the anterior part of the labyrinth-wall adjacent to the jugular vein and the internal carotid artery are seldom affected.

Ulcerative ostitis is thus a commoner result than **necrosis** *en masse*, though the latter often takes place rapidly in children in connexion with the acute exanthemata, and with scarlet fever in particular. Superficial necrosis is most apt to destroy the outer lamella of the promontory beneath the fenestra ovalis, and the cortex of the mastoid process or that portion of it which goes to form the external meatus. Deeper and more extensive necrosis occasionally destroys large portions of the temporal bone, the entire mastoid process with the adjacent parts of the meatus, the promontory with the aqueductus Fallopii and the foramen ovale, the cochlea with some or all of its convolutions, or the semicircular canals: cases have indeed been recorded in which the entire labyrinth became exfoliated and extruded through the external meatus, or was found detached *post mortem*.

The extrusion of such sequestra leaves behind large cavities, which are presently filled up in part or entirely with granulations and osteophytes: in the mastoid process in particular large and deeply-retracted scars or cavities with a thin smooth lining are produced.

Caries of the auditory ossicles may be associated with carious affections of the tympanum or arise independently. The process is usually consecutive to disease of the mucous membrane, but primary ostitis of the ossicles has also been observed. The head of the malleus is oftener eroded than the handle; the body of the incus rarely; while the head and crura of the stapes are often destroyed, leaving the base intact. Extrusion of the entire necrotic malleus or incus during life has repeatedly been observed, but the stapes is rarely thus exfoliated.

Primary **periostitis** of the mastoid process is not very common; it gives rise to superficial necrosis and may lead to perforation outwards or into the auditory canal. An abscess of the lymph-glands overlying the mastoid process, opening by a fistulous sinus, sometimes simulates the features of mastoid periostitis.

Chronic inflammation of the mucous membrane of the middle ear often induces hyperplastic proliferation in the periosteum and medulla of the underlying bone, resulting in **hyperostosis** and the production of **exostoses**. These latter appear as broad-based rounded elevations on the promontory near the fenestra rotunda, and on the pyramidal eminence; they occur much less frequently about the tympanic orifice of the eustachian tube and on the ossicles. By such bony overgrowths the fenestrae of the labyrinth are often obstructed, and the tympanum and eustachian tube contracted. The substance of the mastoid process may become sclerotic, the resulting condensation being in some cases so extreme that the mastoid cells are filled up with compact bone; but the antrum, a cavity of the size of a cherry-stone situated just behind the tympanum, usually persists.

The detection of the tubercle-bacillus in granulations removed by operation from the bone has demonstrated the tuberculous nature of certain forms of mastoid caries; the existence of primary tuberculosis of the temporal bone, such as occurs in other bones of the skull, is still *sub judice*.

As they are occasionally mistaken for the effects of caries, it is well to bear in mind that anomalous lacunae or dehiscences, from defective ossification, are not uncommon in the temporal bone: they are especially frequent in and about the tegmen tympani, but are also met with in the floor of the tympanum, the cortex of the mastoid process, and in the carotid canal. These lacunae are due to physiological resorption, and they have some importance in that they facilitate the extension of inflammatory processes from the tympanic cavity to the adjacent parts.

The petrous bone is occasionally found to be 'pneumatic,' in other words it is pervaded by abnormal air-containing spaces or 'cells.' These surround the labyrinth on all sides, are often of considerable size, and communicate with the mastoid cells. Sometimes they extend down to the very tip of the petrous bone.

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GRUBER: *Monatsschr. f. Ohrenheilk.* XIII **1879**
HABERMANN: *Prager Z. f. Heilk.* VI **1880**, and *A. f. Ohrenheilk.* XXVIII **1890**
HESSLER: *Die otogene Pyämie* Jena **1896**
KANTHACK: *Z. f. Ohrenheilk.* XXI **1892**
KIRCHNER: *V. A.* 41 **1868**
MOOS: *D. med. Woch.* **1888**, and *Z. f. Ohrenheilk.* XX **1891**
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SCHEIBE: *Z. f. Ohrenheilk.* XIX **1890**
SCHÜTZ: Tuberculosis *V. A.* 66 **1876**
SCHWARTZE: *A. f. Ohrenheilk.* II **1867**
VON TRÖLTSCH: *Gesamm. Beiträge* Leipzig **1883**
WEICHSELBAUM: *Monatsschr. f. Ohrenheilk.* XXII **1888**
ZAUFAL: *Prager med. Woch.* **1887**, **1888**, and **1889**

380. **Adhesive processes** resulting from inflammation within the tympanic cavity give rise to a series of changes which deserve special mention. These consist in the formation of membranes, bands, and strings, that traverse the tympanum in various directions, connecting the tympanic membrane, the ossicles, and the tendon of the tensor tympani, with one another or with the walls of the tympanum. More or less extensive adhesions between the tympanic membrane and the labyrinth-wall now and then produce almost complete obliteration of the tympanic cavity.

These filiform and membranous adhesions are referable to the agglutination of the swollen and proliferous mucous surfaces, which is facilitated if the cavity is exceptionally contracted. They consist of loose connective tissue pervaded by tenser fibrous strands, containing few capillaries, and overlaid with flattened epithelium. In some places they are transformed by calcareous infiltration into firm rigid trabeculae.

Of greater importance is the so-called **sclerosis** of the tympanic mucosa and of the articulations of the ossicles. The minute textural changes involved are but imperfectly understood; but they appear in some cases at least to consist in cicatricial contraction of tissue that has previously been relaxed, infiltrated, and hyperaemic. In a few instances sclerotic changes have been detected in the deeper or periosteal layer of the submucosa: in others the submucosa was infiltrated with calcareous granules. Such changes are either generally diffused or limited mainly to special parts of the tympanum, such as the tympanic membrane, the promontory, the fenestrae, and the ossicles.

Adhesions and scleroses of the kind described, even when confined to the mucous membrane, will naturally hamper the movements of the ossicles; but when such changes involve the articular structures also, the relative mobility of these bones must be completely abolished. Fixation from this cause takes place most frequently at the articulation of the stapes with the fenestra ovalis, less often at that between the incus and the malleus.

Ankylosis of the stapes may be congenital, or acquired at any age, but chiefly in advanced life. It depends, in different cases, on calcification of the annular ligament which connects the base of the stapes to the fenestra ovalis, on outgrowths from the cartilaginous investment of the rim of the fenestra, or on hyperostosis of the vestibular surface of the stapes or the rim of the fenestra itself. In one case of ankylosis of the malleus and incus POLITZER found on minute examination that ossification of the articular cartilages of both bones had taken place, and that each had become fused with the ossified interarticular cartilage. That such ankylosis may be due to primary affections of the articular cartilage seems not improbable, though it cannot as yet be regarded as proven.

Such membranes and bands as have been mentioned above are not always the result of inflammation; in many cases they represent the persistent remains of the mucoid tissue that occupies the tympanic cavity in the foetus and new-born infant.

References on Adhesive Processes in the Middle Ear.

MOOS: *A. f. Ohrenheilk.* II **1867**, and *A. f. Augen- und Ohren-heilk.* II **1872**, III **1874**, and VII **1878**
POLITZER: *A. f. Ohrenheilk.* VII **1873** and XI **1876**, and *Lehrbuch* (trans. by CASSELLS) London **1883**
VON TRÖLTSCH: *V. A.* 17 **1859**
WENDT: *A. d. Heilk.* XI-XV **1870-74**

381. Ulceration of the **eustachian tube** at its pharyngeal orifice and in the adjoining cartilaginous portion is the chief morbid change to which it is subject. The ulcers are of various kinds, erosive, follicular, variolous, diphtheritic, syphilitic, and tuberculous, the two last-named occasionally laying bare the cartilage and destroying the greater part of the prominent margin of the pharyngeal orifice. Deep ulcers, such as those due to syphilis, when they cicatrise, and the soft palate at the same time becomes adherent to the posterior wall of the pharynx, are apt to give rise to **stricture** or occlusion of the orifice. Adhesion and stricture of the tympanic orifice sometimes follow upon suppurative caries of the tympanic bones. Complete obliteration of the entire tube is extremely rare, though adhesive bands and membranes are not infrequently found traversing its lumen.

Stenosis and distortion of the pharyngeal opening are moreover liable to result from various morbid conditions of the naso-pharynx, such as acute and chronic swelling of the mucous membrane, hyperplasia of the lymphadenoid tissue in the roof of the pharynx and about the opening of the tube, new-growths, swelling of the posterior end of the inferior turbinate bone, thickening of the velum palati, and cicatrices in the lateral walls of the pharynx. Hyperostoses and exostoses, and undue prominence of the carotid canal, occasionally cause constriction of the osseous portion of the tube.

Among the degenerative changes affecting the tube-cartilage may be mentioned softening and splitting: calcification and disseminated ossification have been described as following upon chronic inflammatory affections of the middle ear.

The internal muscles of the ear undergo fatty or fibroid degeneration in connexion with chronic otitis media and with ankylosis of the ossicles.

The tendon of the tensor tympani is often considerably shortened owing to adhesions with its sheath, or to retraction of the mucous membrane surrounding it.

Similar degenerative changes not uncommonly take place in the tubo-palatine muscles as a result of chronic retro-nasal and tubal catarrh. Hypertrophy of the tensor veli palati has been observed in connexion with the latter affection.

References on the Morbid Anatomy of the Eustachian Tube.

FLESCH: *Tagebl. d. Naturforscherversamm.* Freiburg i. B. **1883**
LINDENBAUM: *A. f. Ohrenheilk.* I **1864**
MOOS: *Norm. u. path. Anat. d. eustach. Röhre* Wiesbaden **1874**; *A. of Ophth. and Otol.* V **1876**
RÜDINGER: *Monatsschr. f. Ohrenheilk.* I–III **1867–69**
WENDT and WAGNER: Diseases of the naso-pharnyx and pharnyx *Ziemssen's Cyclop.* VII New York **1876**

382. The hyperplastic growths and tumours, known clinically as **aural polypi**, are usually found to spring from the middle ear.

Even when they apparently grow from the skin of the bony external meatus they are often found on careful anatomical investigation to originate in one of the cavities that lie outside the canal and are lined with mucous membrane continuous with that of the tympanum. Polypi may arise from any portion of the middle ear, but the inner wall and the roof of the tympanum are the commonest seats, and the tympanic membrane somewhat less frequently. In the mastoid and in the mucous membrane of the eustachian tubes polypi have also been observed. They usually occur in connexion with chronic suppuration accompanied by perforation of the tympanic membrane, and but seldom with non-perforating inflammation.

The growths appear as globular or flask-shaped tumours, provided with slender pedicles or broadly sessile, the surface being smooth or beset with papillary excrescences. A polypus sometimes attains such large dimensions as completely to fill the tympanic cavity and external meatus, and even to protrude as a pyriform mass from the outer orifice of the latter. Such growths are of consequence chiefly from the fact that while they keep up suppuration they obstruct the free discharge of pus, and so expose the patient to the dangers of retention within the tympanum. We have already mentioned the occasional extrusion of considerable fragments of polypous growths as giving rise to severe haemorrhage.

According to their structure polypi are distinguished as granulomatous, fibromatous, and angiofibromatous (MOOS and STEINBRÜGGE), angiomatous, myxomatous, and mucous or myxadenomatous. Mucous polypi are vascular, usually lobulated, soft tumours, resembling the mucous membrane in structure, but richer in cells and enclosing tubular glands and cysts. Angiomatous and purely myxomatous polypi are rare; the latter are said to arise from persistent remnants of the mucoid tissue that normally occupies the foetal tympanum.

The surface is covered with ciliated epithelium or with stratified columnar or squamous epithelium. Mechanical conditions (such as pressure or the friction accompanying extrusion) induce changes in the surface epithelial cells, and a single tumour sometimes exhibits a number of different varieties of epithelium side by side.

Aural polypi often contain extravasations of blood and blood-pigment, and in the centre there are occasionally cholesteatomatous deposits, due to cornification of the epithelial ingrowths from the surface. Internal ossification of the polypous tissue is very uncommon.

Exostoses produced in connexion with chronic inflammation have already been mentioned (Art. 379). Sarcoma, osteosarcoma, and primary carcinoma are very rare; they either give rise to the symptoms of caries, and are at first liable to be mistaken for masses of granulation-tissue or polypi, or they appear as periosteal swellings about the mastoid process. As they grow they destroy the invaded structures, and generally end by involving the entire temporal bone.

Cholesteatoma is a somewhat frequent growth in the middle ear, taking the form of concentrically-stratified masses with a pearly lustre, that either occupy the tympanic spaces as rounded balls, or line its walls with layers of varying thickness. The growth is made up of large rounded or polygonal squamous epithelial (epidermoid) cells, with a few resembling giant-cells, interspersed with numerous crystals of cholesterin: in the centre there is often a nucleus of inspissated pus, but there is no enveloping membrane.

The pearly masses are usually found in the posterior superior portions of the tympanic cavity, in the channel from the cavity into the mastoid antrum, and in the mastoid antrum itself. By pressure on the wall of the tympanum they sometimes erode it and so enlarge its cavity: the entire middle ear is indeed often thereby converted into a single cavernous space occupied by one or more pearly tumours of the size of a walnut.

The epidermoid cells are doubtless derived from the pseudo-epidermal investment of parts of the tympanum by which the mucous membrane is replaced as a result of certain chronic inflammations. When the tympanic membrane is destroyed, or after perforation becomes adherent by its ruptured edges to the wall of the middle ear (Habermann), the cuticular lining of the meatus is apt to become proliferous and grow into the tympanum, whose wall even as far as the mastoid antrum becomes at length invested with epidermis consisting of a rete Malpighii and a stratum corneum. The horny cells of this latter layer are constantly undergoing desquamation, and so become collected and agglutinated within the tympanum and its accessory cavities.

Virchow regards tympanic cholesteatomata as independent heterologous structures analogous to those occasionally met with in the pia mater. Buhl, Kuhn, and Küster refer them to some embryonic anomaly: the latter authority indeed would describe them as 'branchiogenous cystomata' of the petrous bone.

Moos and Steinbrügge deny that true myxadenomatous or mucous polypi are ever met with in the middle ear: they regard the structures hitherto considered to be glandular tubules as produced by ingrowth of the epithelium and by partial cohesion between adjacent portions.

On the mucoid substance occupying the foetal tympanum, and its medicolegal bearings, see von Tröltsch: *Surgical diseases of the ear* (trans. by Hinton) London **1874**.

References on Polypi of the Middle Ear.

Billroth: *Ueber den Bau der Schleimpolypen* Berlin **1855**
Eitelberg: *Z. f. Ohrenheilk.* xvi **1887**
Kessel: *A. f. Ohrenheilk.* iv **1869**

Kiesselbach: *Monatsschr. f. Ohrenheilk.* xvi **1882**
Lucae: *V. A.* 29 **1864**
Moos and Steinbrügge: *Z. f. Ohrenheilk.* and *A. of Otol.* iv **1875**
von Tröltsch: *V. A.* 17 **1859**, and *A. f. Ohrenheilk.* iv **1869**
Zaufal: *Prag. med. Woch.* i **1876**

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Delstanche: *A. f. Ohrenheilk.* xv **1879**
Fränkel: *A. f. Ohrenheilk.* viii **1873**
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Haug: Melanotic giant-celled sarcoma *Ziegler's Beiträge* xvi **1894**
Kipp: Epithelioma *Z. f. Ohrenheilk.* xi **1881** and *A. of Otol.* x **1881**
Lucae: *A. f. Ohrenheilk.* xiv **1878**
Politzer: *Wien. med. Blätter* v **1882**
Schwartze: Epithelioma *A. f. Ohrenheilk.* ix **1875** (with references)

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Bezold: *Z. f. Ohrenheilk.* xx and xxi **1891–92**
Gruber: *Allg. Wien. med. Zeit.* vii **1862**
Habermann: *A. f. Ohrenheilk.* xxvii **1890**; *Prager Z. f. Heilk.* xi **1885**
Haug: *Cent. f. allg. Path.* **1895**
Küster: *Berl. klin. Woch.* **1889**
Kuhn: *A. f. Ohrenheilk.* xxvi **1888**, and *Z. f. Ohrenheilk.* xxi **1892**
Lucae: *Verh. der Berlin. med. Gesell.* i, and *A. f. Ohrenheilk.* vii **1873**
Moos: *A. f. Heilk.* viii **1873** and xi **1876**
Schwartze: *A. f. Ohrenheilk.* ix **1875**
von Tröltsch: *A. f. Ohrenheilk.* iv **1869**, *Lehrbuch* (trans. by Hinton) London **1874**
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CHAPTER CXXI

THE INTERNAL EAR

383. **Anaemia** of the parts of the labyrinth supplied by the auditory branch of the basilar artery is brought about by such changes in this vessel as interfere with the circulation through it, and in particular by chronic endarteritis, tumours compressing it, and basilar aneurysms. In a case of sudden deafness FRIEDREICH was able to show that embolism of the auditory artery had taken place.

Hyperaemia of the labyrinth, assuming the form of a reticulate injection of the vessels or of diffuse reddening, and minute ecchymotic **haemorrhages**, either limited to special parts like the cochlea and vestibule or uniformly diffused, occur in connexion with intense suppurative otitis media with or without caries, especially in the form consecutive to scarlatina. Hyperaemia accompanied by small ecchymoses has been shown to exist in a number of febrile affections, such as typhoid, small-pox, acute tuberculosis, pyaemia, and puerperal fever, as also in mumps (TOYNBEE). In intracranial affections like suppurative or tuberculous meningitis and haemorrhagic pachymeningitis, and in general diseases like leukaemia and pernicious anaemia, the presence of hyperaemia of the labyrinth has repeatedly been demonstrated.

Passive hyperaemia of the labyrinth is apt to accompany general engorgement of the vessels of the head, due for example to the pressure of a goitre or to cardiac and pulmonary affections; but it is sometimes induced by local impediments to the venous efflux, to tumours at the base of the brain, or to thrombosis of the lateral or petrosal sinus.

Copious extravasation into the osseous and the membranous labyrinth is commonly a result of traumatic injury, such as fracture or fissure of the petrous bone, and violent concussion unaccompanied by fracture.

Small haemorrhages may be completely re-absorbed without leaving any permanent disorder of function: in the case of frequently-repeated and of more copious haemorrhages the re-absorption is incomplete, the effused blood remaining as a pigmented deposit. The deposit is apt to induce inflammation, and so lead to atrophy and degeneration of the fibrous and the nervous structures. Purulent disintegration of the effused blood, followed by extension of the suppuration into the cranial cavity, has been observed to take place as a result of traumatic injury.

Inflammation of the labyrinth (*otitis interna*) appears to be very rare as a primary and independent affection; in most cases it is set up by the extension of tympanic or intracranial inflammation to the internal ear. From the middle ear the exciting cause is conveyed by way of the vascular anastomoses in the inner wall of the tympanum, or through erosions of the bone and perforations of the fenestral membranes. In the inflamed labyrinth the blood-vessels may be simply injected or the tissues may be slightly infiltrated with leucocytes, as in typhoid, scarlet fever, and small-pox; but in severe cases the entire membranous labyrinth undergoes suppuration and becomes disorganised. The inflammation then occasionally travels along the neurilemma of the auditory nerve into the cranial cavity, or the pus within the labyrinth becomes inspissated and caseous, while fibrous adhesions block up the internal auditory meatus and so put a stop to the further extension of the disease.

Of the various inflammatory conditions affecting the cranial cavity, epidemic cerebro-spinal meningitis is that which is most apt to give rise to inflammation of the labyrinth: the extension takes place along the trunk of the auditory nerve (HELLER), through the aqueductus cochleae (HABERMANN), or by both routes simultaneously (STEINBRÜGGE).

In the early stages the changes in this form of **internal otitis** consist of hyperaemia and effusion of blood into the labyrinth, with some disorder of the membranous structures; by and by these latter are completely destroyed, the bony walls are denuded of periosteum and eroded, and the labyrinthine spaces are filled with pus and with granulation-tissue which is partially converted into fibrous tissue or even into bone. The inflammatory changes are most pronounced in the vestibule and semicircular canals, and are less perceptible about the cochlea; in the cochlea itself the first or basal coil is generally more affected than the upper ones, the inflammation gradually diminishing as we ascend to the apex.

Inflammations of the labyrinth consecutive to infective diseases such as diphtheria and measles have been described by MOOS. He attributed them to bacterial invasion, and demonstrated the presence of streptococci in the endolymphatic and perilymphatic spaces, in erosions of the trunk of the auditory nerve, and in the periosteum and medullary spaces of the petrous bone. The morbid appearances corresponded generally with those met with in cases of cerebro-spinal meningitis.

So far as can be judged from the scanty records at our disposal, **syphilitic inflammation** of the labyrinth appears to start in the periosteum, and like the other forms issues in the production of new fibrous tissue and of bone, with the result that the membranous structures are destroyed. In leukaemia also, in addition to signs of recent haemorrhage, evidences of similar periosteal inflammation and proliferation have been detected.

Necrosis of the labyrinth is referred to in Art. 379.

In addition to the changes above mentioned as characteristic of internal otitis, such as the production of new fibrous tissue and bone, degeneration and atrophy of the membranous structures and of the nerves, and accumulations of pigment, cholesterin, detritus, and calcareous matter, have been noted in particular cases.

In the well-known case of sudden deafness and abiding **vertigo** described by Ménière, where the only morbid change discovered to account for the symptoms was a sanguineous effusion (*exsudation sanguine*) in the semicircular canals and vestibule, it is still a question whether the essential lesion was haemorrhage or inflammation.

Voltolini strongly maintains that primary inflammation of the labyrinth is the cause of a form of sudden deafness common in children and accompanied by certain definite clinical symptoms, but no pathological evidence of an objective nature has yet been adduced in support of the statement.

Baratoux has succeeded in inducing haemorrhage into the labyrinth by unilateral section of the medulla oblongata in animals. Kirchner's experiments, in which labyrinthine hyperaemia and extravasation appeared to result from the administration of quinine and salicylic acid, are regarded as vitiated by faulty conditions.

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384. Morbid affections of the **auditory nerve**, such as hyperaemia and haemorrhage, are usually associated with corresponding conditions in the labyrinth. **Neuritis** has been observed in association with caries of the petrous bone and cerebro-spinal meningitis, and in cases of traumatic injury such as fracture or fissure of the bone.

Atrophy of the auditory nerve and its branches takes place in connexion with cerebral lesions affecting its place of emergence from the brain, and with apoplexy or encephalitis of the floor of the fourth ventricle, the cerebellum, or the medulla oblongata. Atrophy from pressure is sometimes induced by internal hydrocephalus, tumours of the brain and of the base of the skull, fractures of the petrous bone, and by hyperostoses constricting the internal auditory meatus. Haemorrhage and inflammation affecting the nerve may likewise result in atrophy.

The form of atrophy resulting from disuse, which was once thought to be an invariable result of long-continued peripheral deafness from ankylosis of the stapes with the margin of the fenestra rotunda, seems in fact to be somewhat rare; and even in congenital deaf-mutes the nerve-trunk is often little if at all affected. The peripheral ramifications of the nerve in the labyrinth and in the ganglionic layers of the cochlea, on the other hand, not infrequently become atrophic. In a case described by Moos and Steinbrügge the atrophy was limited to the first coil of the cochlea, in accord with the fact that during life there was deafness to tones of high pitch. According to Erb, atrophy of the auditory nerve sometimes occurs in tabes dorsalis.

The primary new-growths involving the nerve are fibroma, myxoma, sarcoma, and psammoma. Growths in the neighbouring parts are of course liable to invade the nerve and the labyrinth.

Few cases of demonstrable morbid change sufficient to account for the various auditory disorders associated with many meningeal and cerebral affections have been recorded. In the somewhat infrequent cases of deafness following basal meningitis, such changes have been noted as purulent inflammation of the ependyma and softening of the floor of the fourth ventricle, and purulent infiltration with fatty degeneration and shrinking of the nerve-trunk. In epidemic cerebro-spinal meningitis similar changes, in addition to the above-mentioned purulent inflammation of the labyrinth, accompany and account for the deafness that often results. Unilateral or bilateral deafness, such as occasionally occurs in cases of haemorrhage or softening in the pons and cerebellum, and more frequently in connexion with tumours of these regions and with internal hydrocephalus, depends partly on changes in the neighbourhood of the root of the nerve, and partly on traction or compression of its central or peripheral portions. The intermittent deafness observed in cases of acute hydrocephalus is attributable to transient oedema of the auditory centre.

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THOMAS CLIFFORD ALLBUTT,

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Regius Professor of Physic in the University of Cambridge, etc.

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M.A., M.D., LL.D., F.R.C.P., F.R.S., F.L.S., F.S.A.,
Regius Professor of Physic in the University of Cambridge, Fellow of Gonville and Caius College,

AND

W. S. PLAYFAIR,

M.D., LL.D., F.R.C.P.,
Professor of Obstetric Medicine in King's College, and Obstetric Physician to King's College Hospital.

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